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ANALYTICAL STUDY OF 100 CASES OF TRAUMATIC OPTIC NEUROPATHY

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CERTIFICATE

This is to certify that the dissertation entitled, "**ANALYTICAL STUDY OF 100 CASES OF TRAUMATIC OPTIC NEUROPATHY** " submitted by **Dr.V. THAIALNAYAKI** in partial fulfillment for the award of the degree of Master of Surgery in Ophthalmology by The Tamilnadu Dr.M.G.R.Medical University, Chennai is a bonafide record of the work done by her in the Regional Institute of Ophthalmology, Government Ophthalmic Hospital, Egmore, Chennai, during the academic year 2008 – 2011.

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CONTENTS

PART-I

PART-II

PART-III

MASTER CHART

INTRODUCTION

Traumatic optic neuropathy is a devastating potential complication of closed head injury. The hallmark of an optic neuropathy, traumatic loss of visual function, which can manifest by subnormal visual acuity, visual field loss, or colour vision dysfunction. The presence of an afferent pupillary defect strongly suggests a prechiasmal location for the injury and is necessary to validate the diagnosis of traumatic optic neuropathy. Vision loss associated with traumatic optic neuropathy can be partial or complete and temporary or permanent.

Approximately 1.5% to 5% of patients with closed head injuries have damage to visual pathways (4-6 / 100,000 general population / yr). These injuries can be divided into anterior and posterior lesions. Anterior lesions shows ophthalmoscopic abnormalities (eg. Central retinal artery occlusion) and usually associated with a variety of easily recognized injuries to the globe. Anterior lesions may include optic nerve avulsion, traumatic anterior ischemic optic neuropathy, anterior optic nerve sheath hematoma. Posterior lesions are free of ophthalmoscopic findings, but disc edema and disc pallor do occur. Posterior traumatic optic neuropathy is characterised by visual loss that occur in the presence of an afferent pupillary defect but without evidence of injury to the eye or optic nerve.

HISTORY

Hippocrates noted the association of trauma just above the eyebrow and gradual vision loss. By the 18th century, the relationship between frontal trauma and vision loss with an absence of ocular injury was well appreciated. In 1879, Berlin described the first pathologic examination of the optic nerve after head trauma. In 1890, Battle first distinguished penetrating direct from nonpenetrating indirect optic nerve injuries. The 20th century saw significant progress in defining the classification, pathophysiology, and management of traumatic optic nerve injuries.

Historically, observation, medical corticosteroid therapy, or optic canal decompression has been advocated for the treatment of traumatic optic neuropathy. In the early 1900s, transcranial unroofing of the optic canal was the surgical procedure of choice for traumatic optic neuropathy treatment. This procedure was used sparingly because of the inherent risks of intracranial surgery. In the 1920s, Sewell performed a transethmoidal optic canal decompression by removing the lamina papyracea and medial wall of the optic canal. Although his technique was refined by progressive advances in transnasal, transantral, transorbital, and external paranasal sinus surgery, the technique was not performed routinely until the 1960s in Japan and the 1980s in the United

States. During this period, systemic corticosteroid treatment was also extended to treat traumatic optic neuropathy.

Recent advances in endoscopic instrumentation and intranasal sinus surgical techniques have refined extracranial surgical approaches for TON. Currently, endoscopic optic nerve decompression (EOND) via an intranasal and transethmoidal or transsphenoidal approach has gained popular support.

ANATOMY OF THE OPTIC NERVE

WITH BLOOD SUPPLY

EMBRYOLOGY

a) **Optic nerve fibres** develop from the nerve fibre layer of the retina which grow into the optic stalk by passing through the choroidal fissure and pass posteriorly to the brain.

b) **Glial system of the nerve** develops from the neuroectodermal cells forming the outer wall of optic stalk.

c) **The glial septa** surrounding the nerve bundles are composed of astroglia that differentiate from the cells of the inner wall of the optic stalk.

d) **Sheaths of the optic nerve** are formed from the layer of mesenchyme which surrounds the optic stalk [like meninges of other parts of central nervous system], between the 3^{rd} and 7^{th} month of gestation.

e) **Myelination of nerve fibres** begins from the chiasma at about 7^{th} month, proceeds distally and reaches the lamina cribrosa just before birth and stops

ANATOMY OF OPTIC NERVE

Each optic nerve starts from optic disc and extends up to optic chiasma, where the two nerves meet. It consists of axons originating from ganglion cells. It also contains the afferent fibres of light reflex.

PARTS OF OPTIC NERVE

The optic nerve is about 47-50mm in length and can be divided into four parts:

a) Intraocular(1mm)

b) Intraorbital (30mm)

c) Intracanalicular (6-9mm)

d) Intracranial (10mm)

INTRAOCULAR PART

This part passes through the sclera, choroid and finally appears in the eye as optic disc. The intraocular portion of optic nerve head has an average diameter of 1.5mm, which expands to approximately 3mm just behind the sclera, where the neurons acquire a myelin sheath.

The optic nerve head divided into four parts

1. **Surface nerve fibre layer**

   This part is essentially composed of axonal bundles, i.e. nerve fibres of retina (94%) which converge on optic disc and astrocytes (5%).

2. **Prelaminar region**

   The predominant structures at this level are neurons and a significantly increased quantity of astroglial tissue.

3. **Lamina cribrosa region**

   It is a fibrillar sieve-like structure made up of fenestrated sheets of scleral connective tissue lined by glial tissue. It bridges the scleral canal. The bundles of optic nerve fibres leave the eye through these fenestrations. The lamina cribrosa gets its rich blood supply from the circle of Zinn.

4. **Retrolaminar region**

   This area is characterised by a decrease in astrocytes and the acquisition of myelin that is supplied by oligodentrocytes. The addition of myelin sheath nearly doubles the diameter of optic nerve (from 1.5 to 3.0mm) as it passes through the sclera. The axonal bundles are surrounded by connective tissue septa.

INTRAORBITAL PART

This part of optic nerve extends from back of eyeball to optic foramina. This part is slightly sinuous to give play for the eye movements. The relations are as follows:

- The optic nerve in the orbit is covered by dura, arachnoid and pia. The pial sheath sends septa to divide the nerve into fasciculi. The subarachnoid space containing Cerebrospinal fluid ends blindly at the sclera but continues intracranially.
- Central retinal artery and vein crosses the Subarachnoid space to enter the nerve on its inferomedial aspect about 10mm from the eyeball.
- Posteriorly, near the optic foramina, the optic nerve is closely surrounded by the annulus of Zinn and the origin of four recti. Some fibres of superior rectus and medial rectus muscles are adherent to its sheath here.
- Anteriorly, the nerve is separated from the extra ocular muscles by the orbital fat.
- The long and short ciliary nerves and arteries surround the optic nerve before these enter the eyeball.
- Between the nerve and the lateral rectus are situated the ciliary ganglion, divisions of oculomotor nerve, the nasociliary nerve, the sympathetic and the abducent nerve.

- The ophthalmic artery, superior ophthalmic vein and the nasociliary nerve cross the optic nerve superiorly from lateral to medial side.

INTRACANALICULAR PART

- This part is closely related to the ophthalmic artery which crosses the nerve inferiorly from medial to lateral side within the dural sheath.

- Sphenoid and posterior ethmoidal sinuses lie medial to it and are separated by a thin bony lamina.

INTRACRANIAL PART

- This part, about 10 mm in length lies above the cavernous sinus and converges with its fellow (over the diaphragma sellae) to form the chiasma.

- It is ensheathed in pia matter, but receives arachnoid and dural sheaths at its entry into the optic canal.

- The ophthalmic artery arises from the ICA (Internal carotid artery) below the optic nerve at about its middle.

MENINGEAL SHEATHS OF OPTIC NERVE

- The intracranial part of optic nerve is covered by pia only, while the intracanalicular part and intraorbital parts have three coverings: the pia, arachnoid and dura.
- The meningeal sheaths and subarachnoid and subdural spaces around the optic nerve are continuous with those of the brain.
- Anteriorly, all the three meningeal sheaths terminate by becoming continuous with the sclera.
- At the apex of the orbit, the dura split into layers, the outer is continuous with the periosteum of the orbit while the inner forms the dural sheath of the optic nerve.
- The pia sends numerous septa to the optic nerve, dividing its fibres into fascicles.

BLOOD SUPPLY OF THE OPTIC NERVE

ARTERIAL SUPPLY

1. Intra ocular part

a. **Surface nerve fibre layer:** this part is supplied by capillaries from retinal arterioles, which anastomose with vessels of the prelaminar region.

b. **Prelaminar region:** this part is supplied by vessels from peripapillary choroidal system or from separate branches of the short posterior ciliary arteries.

c. **Lamina cribrosa region:** this part is supplied by ciliary vessels from the short posterior ciliary arteries and arterial circle of Zinn-Haller.

d. **Retrolaminar region** is supplied by both ciliary and retinal circulation, the former coming from recurrent pial vessels and the later from pial plexus of CRA (central retinal artery).

2. **INTRAORBITAL PART**

a) **Periaxial system** of vessels derived from the six branches of ICA namely: ophthalmic A, long posterior ciliary arteries , short posterior ciliary arteries, lacrimal A and central retinal A before it enters the optic nerve and circle of Zinn.

b) **Axial system** of vessels derived from 1.the intraneural branches of the CRA, 2.central collateral arteries which come off from the CRA before it pierces the nerve and 3.central artery of optic nerve.

The two systems have anastomosis between them**.**

3. **INTRACANALICULAR PART**

The nerve within the optic canal is supplied only by the **periaxial system** of vessels and is fed mainly by the ophthalmic A.

4. **INTRACRANIAL PART**

This part of the optic nerve is exclusively supplied by the periaxial system of vessels. The plexus here is contributed by 4 sources: a) branches from ICA either directly or through the recurrent branch of anterior superior hypophysial A, b) branches from anterior cerebral artery c) small recurrent branches from the ophthalmic A and d) the twigs from the anterior communicating A.

VENOUS SUPPLY

- The venous return from the optic nerve head (ONH) is by the Central retinal vein (CRV).
- The orbital part is drained by peripheral pial plexus and also by CRV in the distal part.

The intracranial part is drained by the pial plexus which ends in anterior cerebral vein and basal vein.

OPTIC CANAL

The optic nerve is transmitted to the brain through the optic canal, a foramen of lesser wing of sphenoid. The orbital opening is elliptical with the major axis vertical and minor axis horizontal. The intracranial opening is also elliptical with a 90° rotation in the orientation of axis, so that the horizontal axis is the major axis and the vertical axis is the minor axis. The optic canal is 9.22 mm in length on average. The medial wall of the optic canal is the thinnest. This bone separates the ethmoidal air cells and sphenoid sinus.

The canal transmits the optic nerve with its dural sheath, the ophthalmic artery and the fibres of sympathetic carotid plexus. Unlike any other portion of optic nerve, the intracanalicular part is tightly bound. Within the canal the optic nerve dura fuses with the periosteum of the optic canal.

ETIOLOGY

Traumatic optic neuropathy (TON) is most commonly caused by **motor vehicle and bicycle accidents** (15-75% of cases, depending on the series). **Falls** (15-50% of cases) are the next most common cause, followed by **physical violence** and **recreational sports.**

Direct traumatic optic neuropathy is the term used when the optic nerve is impinged, crushed, or transected. These injuries are usually the result of open craniofacial trauma, such as penetrating wounds (eg, from knives) or extensive crush injury with displaced cranio-orbital fractures.

Indirect traumatic optic neuropathy occurs in the absence of direct optic nerve injury and is more common than direct traumatic optic neuropathy.

Blunt trauma classically occurs following rapid decelation injuries to the anterofrontal regions of the head. Trauma to the outer third of the superior orbital rim is transmitted directly to the optic canal where the optic nerve is tethered at both ends by the dura.

Severity of the trauma does not always correlate with the degree of visual loss. The incidences such as minor fall after tripping or hitting the side of the head against a solid object resulting in a frontal blow are adequate to produce a posterior traumatic optic neuropathy.

The presence or severity of orbital fractures neither directly predicts the severity of visual loss nor determines prognosis.TON may be associated with the fracture of the medial wall, optic canal, zygoma or floor. Patients with optic canal fracture may regain normal vision without intervention and those with no fracture may present with no light perception vision that persists despite all interventions.

TON is most often seen in **boys** in their **first or second decade** of life. Patients older than 40 yrs of age were found to have worst visual outcome independent of mechanism of injury, severity of visual loss, or intervention utilized.

CLASSIFICATION OF OPTIC NERVE INJURURIES

Anatomical classification

Anatomically it can be classified as,

1. Optic disc trauma (avulsion)
2. Anterior optic neuropathy
3. Posterior optic neuropathy.

Optic disc trauma

Avulsion of the optic nerve as it enters the globe produce a partial ring of haemorrhage at the optic nerve head. Avulsion site may be visible as a crescentic dark area at the disc.

Anterior optic neuropathy

Injury to the proximal portion of the optic nerve within 10 mm of the globe, anterior to where the central retinal artery enters and the central retinal vein leaves nerve produce a central retinal or branch retinal artery occlusion or anterior ischemic optic neuropathy.

Posterior optic neuropathy

Injuries to the optic nerve posterior to the entrance and exit of the central retinal artery and vein produce no immediate change in the

appearance of the ocular fundus. Optic disc remains normal for at least three to five weeks, following which it becomes pale. The most common site of posterior indirect optic nerve injury is the optic canal.

The intracranial optic nerve is the next most common site of injury, produce hemianopic field defect. If chiasm is injured it leads to bilateral injury.

It is important to distinguish among injuries to the orbital, intracanalicular and intracranial portions of the optic nerve, because the treatment of injuries to the three different areas is quite different.

The prognosis of an optic nerve injury depends on whether it is direct or indirect. Direct injuries tend to produce severe and immediate visual loss with little like hood of recovery. Indirect injuries are not infrequently associated with visual recovery and also produce delayed visual loss that occurs several hours to days after the injury.

PATHOPHYSIOLOGY

The optic nerve axons lie in two compartments: the intradural and intrafascicular. Closed space edema, contusion necrosis, nerve fibre tears and infarction due to thrombosis or spasm have all been implicated as potential mechanisms of optic nerve injury. Shearing, stretching, compression and contusion at the level of intracanalicular optic nerve are the most important mechanisms in creating optic nerve dysfunction. Surgery can only open the dura and cannot relieve intrafascicular pressure elevations. Interruption of venous flow may also play a major role. Myelin is more sensitive to edema and acidosis than axonal structures. The typical findings found at autopsy include haemorrhage, demylination, focal necrosis and axonal changes.

The exact pathophysiology of traumatic optic neuropathy is poorly understood. Although optic nerve avulsion and transection, optic nerve sheath hematoma, and optic nerve impingement (from a penetrating foreign body or bony fracture) all reflect traumatic mechanisms of the optic nerve dysfunction, these are less common forms of traumatic neuropathic vision loss.

Traumatic optic neuropathy, in its most common form, is an indirect event that occurs during or shortly after blunt trauma to the superior orbital rim, lateral orbital rim, frontal area, or cranium. The most widely held belief maintains that compression forces from the

trauma are transmitted via the orbital bones to the orbital apex and optic canal. Laser interferometry studies demonstrate that forces applied to the frontal bone are concentrated and transferred to the orbital apex and anterior foramen of the optic canal. Elastic deformation of the sphenoid then allows transfer of the force to the intracanalicular segment of the optic nerve. Contusion of the intracanalicular optic nerve axons and pial microvasculature produces localized optic nerve ischemia and edema. The edematous ischemic axons result in further neural compression within the fixed-diameter bony optic canal, precipitate a positive feedback loop, and trigger the development of an intracanalicular compartment syndrome.

Although ischemia is considered the secondary event that gives rise to the neuropathy, the cellular and subcellular events that constitute the mechanism of neural damage are only now being realized. The roles of oxygen free radicals, enzymes, cytokines, intracellular calcium, and other forms of reperfusion damage are slowly being uncovered through basic science research.

A less common form of traumatic optic neuropathy that involves the intracranial optic nerve results from forces delivered by the brain's shift at the moment of impact. The intracranial optic nerve is sheared as it moves against the falciform dural fold as it overlies the sphenoid plane.

Histologic Findings

Clinicopathologic studies, however, have demonstrated several features of traumatic optic neuropathy, as follows:

- Blood within the optic nerve sheath
- Interstitial optic nerve haemorrhage
- Fibrosis of the pial septa
- Lymphoplasmacytic infiltration
- Iron-laden macrophages
- Triangular-shaped axonal degeneration with loss of myelin
- Ischemic necrosis

The time-dependent histopathologic changes of the optic nerve following indirect trauma have not been adequately described.

NEURO-OPHTHALMIC EVALUATION

The diagnosis of traumatic optic neuropathy is clinical. Patients with midfacial and cranial trauma should elicit a high index of suspicion for traumatic optic neuropathy. Although patients with traumatic optic neuropathy may have serious and obvious craniofacial, neurosurgical, and other co-morbidities, they may also have no visible signs of injury. In addition, although 50% of patients with traumatic optic neuropathy present with a visual acuity of light perception or no light perception, nearly 20% of patients have a visual acuity of 20/200 or better.

Assume optic nerve dysfunction when a loss of best-corrected visual acuity or visual field is accompanied by an ipsilateral afferent papillary defect (APD) (eg, Marcus Gunn pupil). Obtain a detailed medical history and identify premorbid ocular conditions that may limit vision recovery. If the patient's clinical situation limits detailed communication, query the patient's family, paramedics, or witnesses to the trauma about the details of the injury.

Comprehensive ophthalmic examination on all patients in whom traumatic optic neuropathy is suspected is essential and include the following assessments:

- **Ocular adnexa:** Examination may reveal orbital rim and wall fractures, orbital edema, proptosis or enophthalmos, or extraocular muscle dysfunction. Signs of penetrating injuries, such as protruding foreign bodies, extruding orbital contents, or conjunctival laceration, may range from obvious to subtle.

- **Visual acuity:** Assess visual acuity immediately upon presentation. Perform a second assessment within 24 hours of the first to discern cases of delayed optic neuropathy (<10% of traumatic optic neuropathy cases).

- **Pupillary reaction:** An afferent pupillary defect is a necessary condition for the diagnosis of traumatic optic neuropathy. Pupillary reaction is evaluated with the swinging flashlight test (i.e., briskly alternating a flashlight beam from one eye to the other). An afferent pupillary defect is detected when the pupil's initial constriction is replaced by initial dilation.

- **Intraocular pressure:** Increased intraocular pressure may accompany an orbital hematoma, diffuse orbital haemorrhage, orbital emphysema, or soft tissue edema.

- **Ophthalmoscopy:** Perform ophthalmoscopy with the aid of a short-acting mydriatic agent (pupillary dilation) on all stable patients. Evaluate the retinal and choroidal circulation, optic nerve head morphology, and the presence of ring-shaped haemorrhage adjacent to the optic nerve head.

Laboratory Studies

- Haemostasis is essential during optic canal decompression. Obtain the following tests as suggested by the patient's medical history:
    - Hemoglobin/hematocrit
    - Platelet count
    - Prothrombin time (PT)/activated partial thromboplastin time (aPTT)
    - Bleeding time

Imaging Studies

Although most patients with posterior traumatic optic neuropathy have normal imaging studies, CT without contrast should be performed in all cases. Imaging will allow identification of associated fractures, optic nerve avulsion, optic nerve sheath hematoma and optic nerve compression due to orbital hematoma.

Thin-slice CT scan of the nose, sinuses, orbits and optic canal

- CT scanning provides adequate imaging of orbital soft tissue and is better than MRI at delineating bony defects. A thin-section CT scan also provides an intraoperative road map for the surgeon in patients who require surgical decompression.

- The decision for surgical decompression should still be based primarily on the clinical examination findings and not the CT scan findings. Small-review series have concluded that the extent of bony canal injury documented at surgery was underestimated by CT scan findings.
- In polytraumatized patients with poor awareness, CT scan with clinical exploration is the most important method for the assessment of traumatic optic neuropathy in the acute emergency setting.
- **Fractures through the optic canal can be best depicted with thin-section CT scanning (eg, 1.5-mm cuts with 1-mm intervals)**.

- Surgeons who wish to perform image-guided optic canal decompression need to obtain a special-order CT scan that is formatted to their computerized stereotactic localizing system.
- Spiral CT allows rapid data acquisition in uncooperative adults and children.
- MRI is only indicated if intracranial injuries are present that are inadequately detailed with CT imaging.

Other tests

- **Visual field perimetry:** Patients suspected of sustaining traumatic optic neuropathy should undergo visual field testing. Although no visual field defects are pathognomonic of traumatic optic neuropathy, quantification of visual field defects is useful to assess convalescent visual improvements. Simple visual field screening can be accomplished at the bedside for unstable patients, but formally assess patients who can be evaluated in the clinic setting.

- **Multifocal visual-evoked potential (VEP), multifocal electroretinography (mfERG), and optical coherence tomography** are 3 promising techniques in the future diagnoses of subclinical vision loss. Some of these tests are already used in neuroophthalmology for the studies of the retina and glaucoma. Although none of these techniques should replace a careful history and clinical examination, these techniques might be important as adjunct procedures in the **evaluation of the unconscious patient or patients with bilateral optic neuropathy**. Flash visual-evoked potential (FVEP) was studied in patients with traumatic optic neuropathy with calculation of a ratio of the amplitude of the injured to the uninjured eye. A ratio of greater than 50% was associated with favourable visual outcome.

NATURAL HISTORY

The natural history of traumatic optic neuropathy is difficult to characterize because each patient is different. Attempts to study patient outcomes have also been hindered by the assumption that corticosteroids are helpful and that not offering them would be unethical. Visual prognosis and likelihood of spontaneous improvement are independent of the initial visual acuity.

The natural history of indirect optic nerve injuries has been described in several clinical series. The rate of spontaneous visual improvement ranges from 20% to 57% as shown in following table:

NATURAL HISTORY OF TRAUMATIC OPTIC NEUROPATHY

Author(Year of Study)	Number of patients	Spontaneous improvement (%)
Tang[14] (1986)	13	38
Millesi[13] (1988)	07	57
Lessell[14] (1989)	25	20
Seiff [7] (1990)	15	33
Levin [6] (1999)	09	57

Even patients who had a return of normal central visual acuity did not regain an entirely normal afferent examination. Persistence of visual

field and colour defects, and APD are typical. Optic nerve pallor or nerve fibre layer changes also develop over the months following the injury. Patients with no light perception (NLP) on presentation can sometimes recover useful vision without intervention.

The prognosis tends to be better for patients who have a lucid interval or an enlarged nerve sheath but poorer for patients older than 40 years. The following factors do not appear to correlate with visual outcome.

- Gender
- Level of consciousness
- Mechanism of injury
- Initial visual acuity (including NLP)
- Presence of fractures (including optic canal fracture), and
- Time from injury to intervention (within the first 7 d).

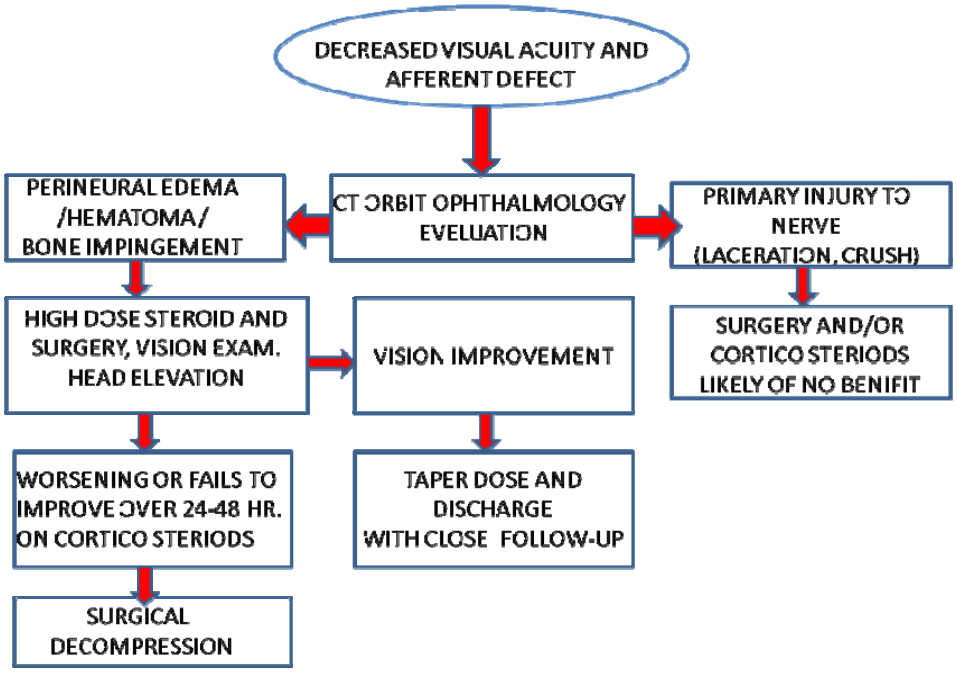
DIFFERENTIAL DIAGNOSIS

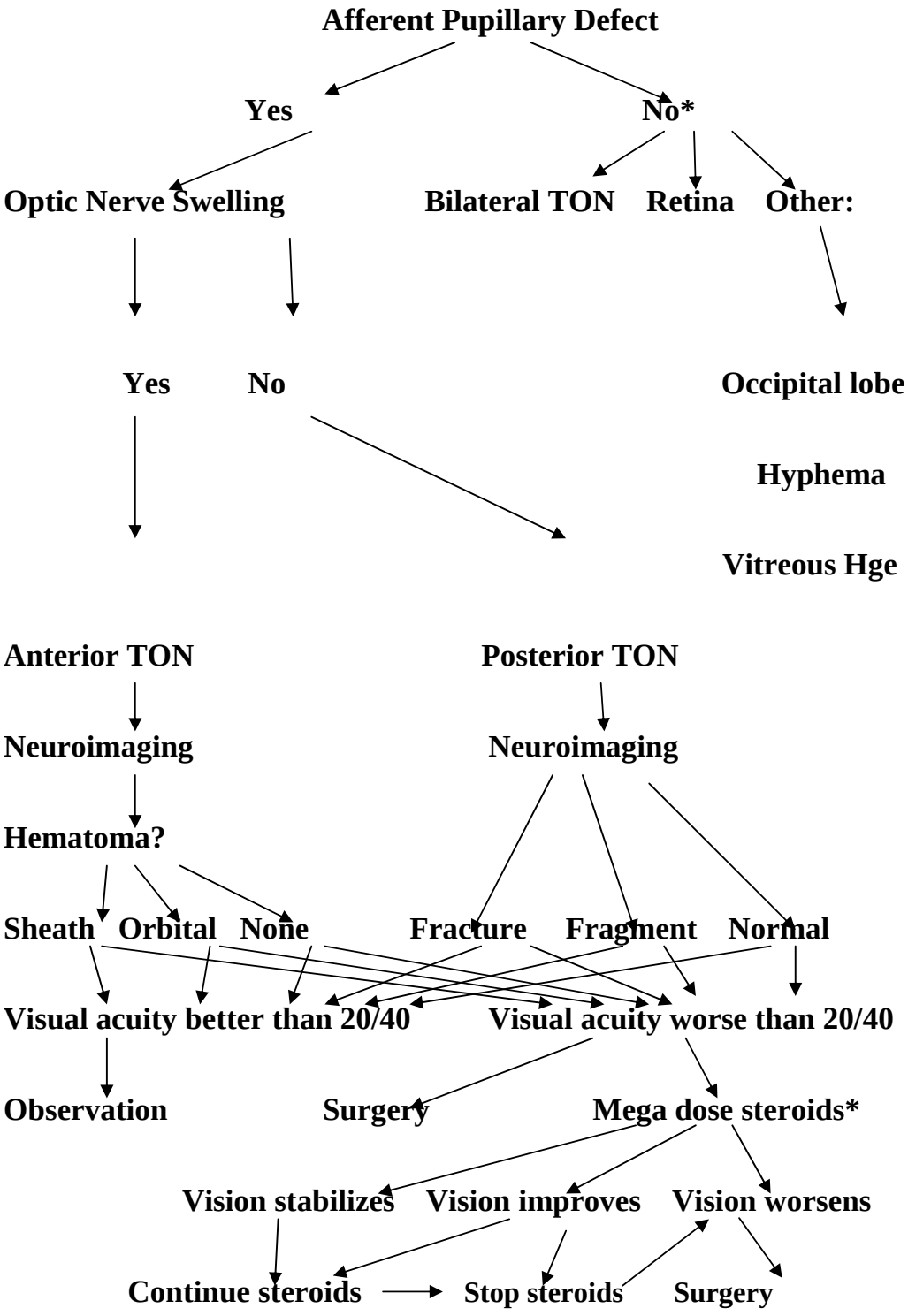
With a thorough history and complete clinical examination, the differential diagnosis of visual loss with disturbed pupillary function can be narrowed.

Monocular visual loss may not be noticed until the individual has occasion to close the uninvolved eye. In this setting patient may attribute the visual loss to a recent traumatic event. Deciding the visual loss is co-incidental or causal may be difficult. Any process that can result in visual loss may be co-incidental with a traumatic incident such as decomposition of a vascular aneurysm, orbital or optic nerve inflammation, anterior ischemic optic neuropathy or acute sinus disease with orbital involvement. A traumatic cavernous sinus fistula is usually accompanied by numerous orbital findings permitting easy differentiation from traumatic optic neuropathy.

TREATMENT

During the mid 1970s, observation was replaced by steroid therapy and surgery as a variety of case series promoted the success rates of these improvement without treatment has been well documented. Despite the rate of spontaneous improvement, however, the standard of care has been to offer high dose corticosteroids. The role of surgical intervention is still debated. The International Optic Nerve Trauma Study (published in 1999) demonstrated no clear benefit from either corticosteroid therapy or optic canal decompression surgery. Observation has again become an acceptable option when there is no evidence of intrasheath hematoma, orbital hematoma, or optic canal fracture fragments.





***Loading dose: I.V. Methyl prednisolone 30 mg/kg followed by 15 mg/kg 2 h later. The maintenance dose could then be 15 mg/kg at 6 h intervals or 5.4mg/kg/h for at least 48 h before deciding whether response has occurred. TON-traumatic optic neuropathy**

Corticosteroid Therapy

Widely used for several decades, corticosteroids are thought to stabilize lipid membranes, reduce spasm, increase blood supply, and reduce neural tissue edema and necrosis. Mega dose steroids were first studied in animal models with brain edema and then in humans with spinal cord injuries. Spoor[11] and colleagues were the first to use spinal cord doses for patients with traumatic optic neuropathy. They divided their patients into two groups, and treated

- One group (13 patients) with methyl prednisolone (30 mg/kg initially, then 15 mg/kg given 2 h later and then in divided doses every 6 h) and
- The second group (8 patients) with high dose dexamethasone (20 mg every 6 h)

Both therapies were continued for 48 hours, then followed by a rapid taper. Of the treated eyes (one per patient), 17 of 21 (81%) improved in visual function during these protocols. The relative dose of steroid was much higher for the methylprednisolone group, but the overall outcome was no different. The methylprednisolone group appeared to make a more-rapid recovery, however. A delay in treatment did not appear to alter outcome in this study. The mean onset of therapy was 4.2 days. The authors of the study believed that neither the initial

severity nor the type of nerve injury allowed the response to steroid therapy to be predicted, as five of eight eyes in this study initially had NLP but regained significant function.

Another series by Bendel[20] and colleagues described 17 patients treated with 2 g methylprednisolone initially, then 1 g, divided, every 6 hours for 48 hours. Vision improved in all patients, with an average pre-treatment vision of 20/100 and average post treatment vision of 20/25. Best vision occurred between 1 and 6 days after treatment (mean =2.9 d). Three patients demonstrated deteriorating visual function during steroid taper and were treated with extracranial optic nerve decompression; all three had eventual improvement.

Mauriello[10] and colleagues reported a series in which 9 of 16 patients (56%) treated with a more-conventional steroid dose had significant improvement (1 g loading dose, then 150 mg in divided doses every 6 h for 48 h). Almost all patients who did not improve had NLP vision initially. They also noted that all 5 of their patients with a lucid interval following injury had eventual improvement.

In summary, a short course of high-dose steroid may be considered unless there is clear evidence of optic nerve transection or avulsion by clinical or radiographic criteria. A delay in onset of therapy and the degree of visual loss have not been clearly shown to alter prognosis as evidenced by following table.

REPORTED OUTCOMES USING CORTICOSTEROIDS

Author(Year of Study)	Number of Patients and (Treatment Received)*	Visual improvement (%)
Tang[14] (1986)	05(C) 11(M)	20 36
Millesi [13](1988)	02(U)	50
Lessel[4](1990)	04(U)	25
Seiff[7] (1990)	21(M)	62
Spoor[11] (1990)	22(M)	86
Bendel[20] (1993)	17(M)	100
Levin[6] (1999)	85(M)	52

*C: conventional steroid dose (1 g methylprednisolone loading dose followed by 250 mg methylprednisolone qid).

M: mega dose steroid doses (30 mg/kg methylprednisolone loading dose followed by 15 mg/kg every 6 h or 5.4 mg/kg/hr for 24—48 hrs).

U: Unspecified.

Surgical intervention

Optic canal decompression was first described in 1916, wherein a transcranial unroofing was performed in patients with afferent dysfunction who required craniotomies for other reasons. Extracranial techniques were later described to minimize the possible complications

associated with craniotomy. A transethmoidal approach was investigated as early as 1926 but not popularized until the 1960s. External, transantral, Caldwell-Luc, and transnasal approached have all been tried. Comparison of different clinical series is difficult because of differences in techniques, selection criteria, and quantification of visual improvement. Many patients also receive at least conventional doses of corticosteroids in the peri-operative period, further confusing the ability to attribute improvement to the surgical intervention. The frequency of reported visual improvement ranges from 12% to 79%. In many medical centres, the decision to operate is governed by the criteria established in 1966 by Walsh

CRITERIA GOVERNING SURGICAL INTERVENTION IN PATIENTS WITH TRAUMATIC OPTIC NERUOPATHY

Absolute Surgical Contraindication

- Optic nerve avulsion is present on CT imaging

Relative Surgical Contraindications

- Patient is unconscious
- Total loss of vision and pupillary response

Relative Surgical Indications

- If visual loss develops despite steroid treatment
- If visual decline occurs during the steroid taper
- If an optic canal fracture is accompanied by potentially compressive bone fragment
- If the visual evoked potential (VEP) response deteriorates over time

Mauriello[10] and colleagues treated 23 patient with steroids and operated on 7 nonresponders based on CT evidence of surgical pathology (optic nerve sheath enlargement or narrowing of the optic canal by bone spicules). Of the 3 patients treated with optic nerve sheath fenestration alone, only 1 had significant improvement. The remaining 4 patients were treated with both fenestration and optic canal decompression. Only 1 patient had significant improvement (NLP to 20/200).

Joseph[30] and colleagues reported visual improvement in 11 of 14 patients treated with steroids and transethmoidal decompression of the optic canal. This report was a retrospective review and not compared to any large, steroid-only treatment group, but the results are similar to

those in the study of Spoor and colleagues, who used mega dose corticosteroids. (described above).

Levin[6] and colleagues reported the outcome of the International Optic Nerve Trauma Study in 1999. The goal of this endeavour was to compare the visual outcome of traumatic optic neuropathy treated with corticosteroids, treated with optic canal decompression surgery, or observed without treatment. Patients who were randomized to the surgical group did not receive steroids in the peri operative period. Intervention occurred within the first 7 days following injury. The main outcome measure was defined as visual acuity improvement of three or more lines of Snellen acuity. The international Optic Nerve Trauma study found that only 32% in the surgery group (n=32) improved significantly. This was in contrast to the visual improvement observed in the untreated group (57%, n=9) and steroid group (52%, n=85). The study concluded that there was no clear benefit for intervention. In addition, they confirmed the findings of others that the timing of corticosteroids or surgery within the 7-day window did not affect outcome.

SUMMARY

Traumatic optic neuropathy is a rare but significant cause of post traumatic visual loss. The responsible blunt trauma to the frontal region may be minor or severe and accompanied by multiple adjacent fractures. Careful documentation of visual acuity, pupillary function, and red desaturation is essential to guide management. CT imaging should be performed to document such structural abnormalities as optic nerve avulsion, optic nerve sheath hematoma, orbital hematoma, or optic canal fracture with fragments.

Based on the data from the International Optic Nerve Trauma Study, observation without intervention is a viable option. Patients and their families should be made aware of the information regarding mega dose corticosteroid therapy and participate in an informed decision. In particular, if visual acuity begins to deteriorate, then corticosteroid therapy should be considered. If a structural abnormality is present that may be contributing to optic nerve dysfunction (hematoma or fragment) or if the patient's visual acuity deteriorates on corticosteroids, optic canal decompression should be offered. Management of this disorder remains very controversial; involvement of other appropriate subspecialists and careful discussions with the patient and family are essential to maximize visual outcome.

FUTURE AND CONTROVERSIES

A better understanding of the cellular and biochemical mechanisms involving normal and traumatized axons, glia, and myelin sheaths may eventually leads to better intervention for traumatic optic neuropathy. Although corticosteroids are a type of neuroprotective agents, the recent expansion in basic scientific research regarding other neuroprotective agents may lead to a redirection in future therapy for this condition.

AIM AND OBJECTIVES

To analyse the clinical profile, response to mega dose steroid therapy and visual function outcome in patients with traumatic optic neuropathy presenting to the neuroophthalmology clinic at regional institute of ophthalmology and government ophthalmic hospital Chennai

MATERIALS AND METHODS

A prospective observational case study on the pattern of traumatic optic neuropathy and analysis of clinical profile, response to treatment and visual function outcome was conducted in the department of neuroophthalmology at RIO-GOH, Chennai.

The study was conducted from June 2008 to November 2010. All eligible traumatic optic neuropathy patients according to inclusion criteria, presented to the neuroophthalmology clinic at RIO-GOH during June 2008 to may 2010 were included in the study. The patients were followed up till November 2010.

INCLUSION CRITERIA

H/o impact to head and orbit

Reduced BCVA in one eye

Relative afferent pupillary defect

Defective colour vision

Field defects

Fundus changes – Normal / Partial / Total optic atrophy

Associated extra ocular muscle palsy

EXCLUSION CRITERIA

Major head injury

Unconscious patients

Eyes with penetrating trauma

Candidates for decompression surgery

Clinical features requiring neurological / neurosurgical interventions.

CLINICAL EVALUATION OF CASES

All cases referred to neuroophthalmology clinic either from general ophthalmic outpatient department, neurological, neurosurgical, general medicine department, who have decreased visual acuity following impact on head / orbit were evaluated in detail. An informed consent was taken from all eligible patients for inclusion in the study.

The patient particular like name, age, sex, address, were documented. A detail history regarding the cause of blindness like time and nature of the trauma, level of consciousness after trauma, site of injury, onset and duration of symptoms and time of presentation, were noted.

The patients were also enquired about h/o systemic illness or surgical instruments if any eg. Sinus or cranial surgeries which could influence the diagnosis.

A patient presented to the neuroophthalmology clinic has to undergo routine

1. visual acuity
2. refraction
3. pupillary reaction
4. slit lamp examination
5. fundus and tonometry examination
6. colour vision
7. field of vision
8. extra ocular movements were checked whenever necessary and possible.

If visual acuity is poor or patient is un co-operative projection field or at least confrontation field, have been assessed.

X – ray skull lateral view, orbit PA view, CT orbit with optic canal axial and coronal, CT brain plain and contrast were taken when found necessary and possible.

Cases were referred to neurology / ENT /orbit department in view of surgical management if required.

Patient’s visual acuity, pupillary reaction, colour vision, visual field and fundus were assessed during follow-up examination at one month, three months, six months and one year.

ANALYSIS

Analysis of the collected data was done based on the following

1. Incidence

2. Age and sex distribution

3. Mode of injury :

   RTA / fall / others

4. Onset of symptoms :

   Immediate (within 24 hours) / 24 hours to one month / late (>1 month)

5. Time of presentation :

   Within one week / 1 week to 1 month / 1 month to 3 month / > 3 month

6. Presenting visual acuity

   a) No PL / PL /HM
   b) 1/60 – 3/60
   c) 4/60 – 6/60
   d) 6/36 – 6/18
   e) 6/12 – 6/6

7. Site of injury:

   Eye brow / fore head / side of face /other parts

   Skull and orbit

8. Fundus status:

   Normal / abnormal

9. Radiological finding:

   Normal / abnormal

10. Treatment:

    Observation / oral steroids / parentral steroids/ surgery

11. Visual acuity at follow up:

    Static / improved / deteriorated.

RESULTS

Based on exclusion and inclusion criteria 100 cases were taken up for the study.

Demography :-

Incidence :-

Total No. of neuroophthalmic cases in the study period	1923
No. of traumatic optic neuropathy cases	100

From the table it can be seen that traumatic optic neuropathy constituted about 5.2 % of the neuroophthalmology cases within the specified period of study.

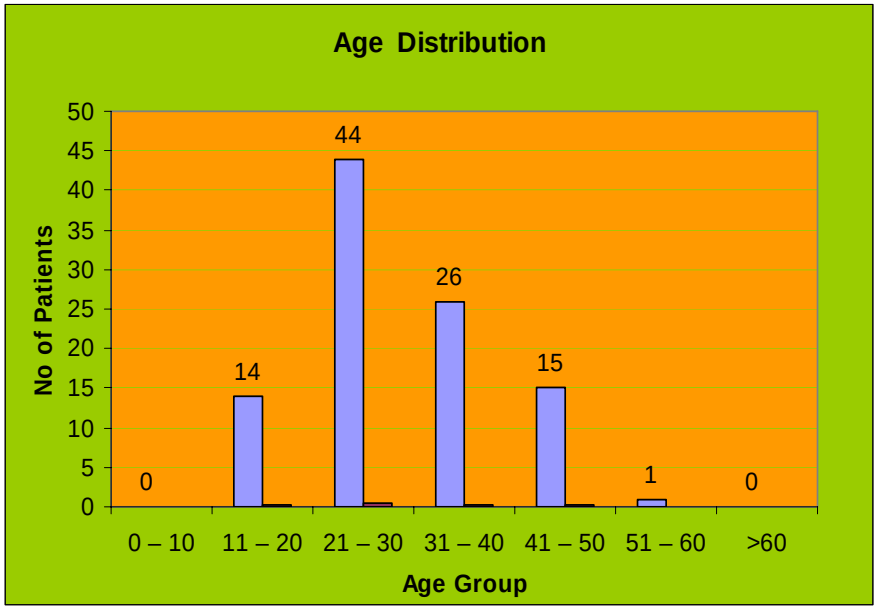
AGE DISTRIBUTION OF CASES

Minimum	**Maximum**	**Mean**
12	52	30.22

AGE GROUP

Age group	Frequency	Percentage
0 – 10	0	0
11 – 20	14	14%
21 – 30	44	44%
31 – 40	26	26%
41 – 50	15	15%
51 – 60	1	1%
>60	0	0

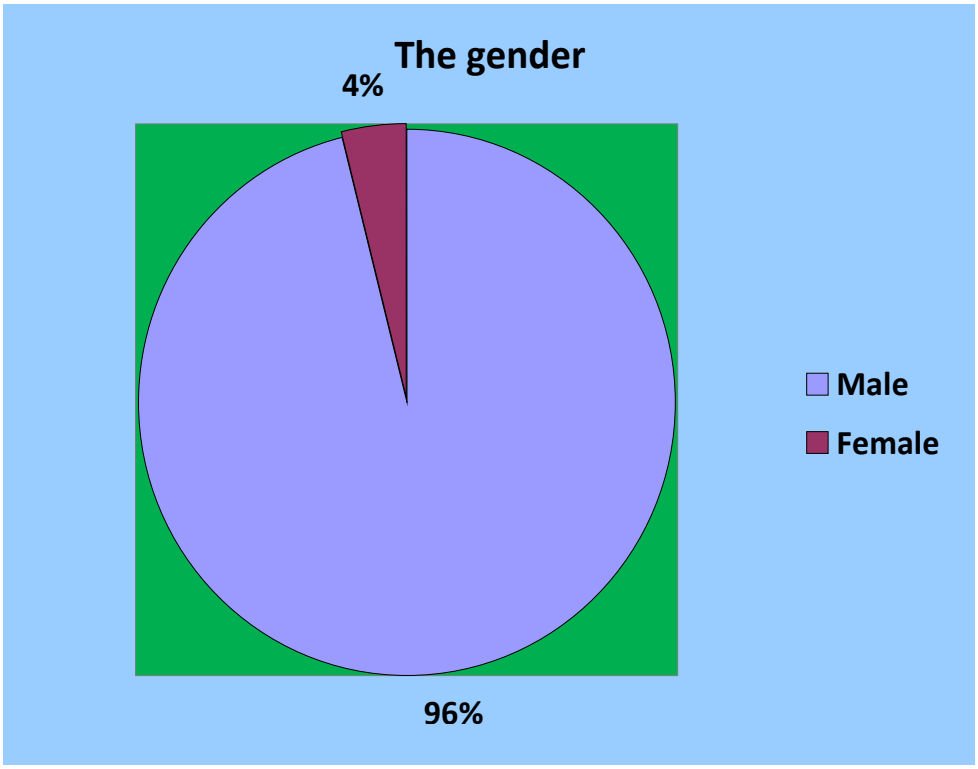
In our study 70% belonged to the age group 20 – 40yrs. The minimum age was 12yrs and the maximum age was 52 yrs. The mean age was 30.22yrs with SD of 9.28.



THE GENDER

Gender	Frequency	Percentage
Male	96	96
Female	4	4

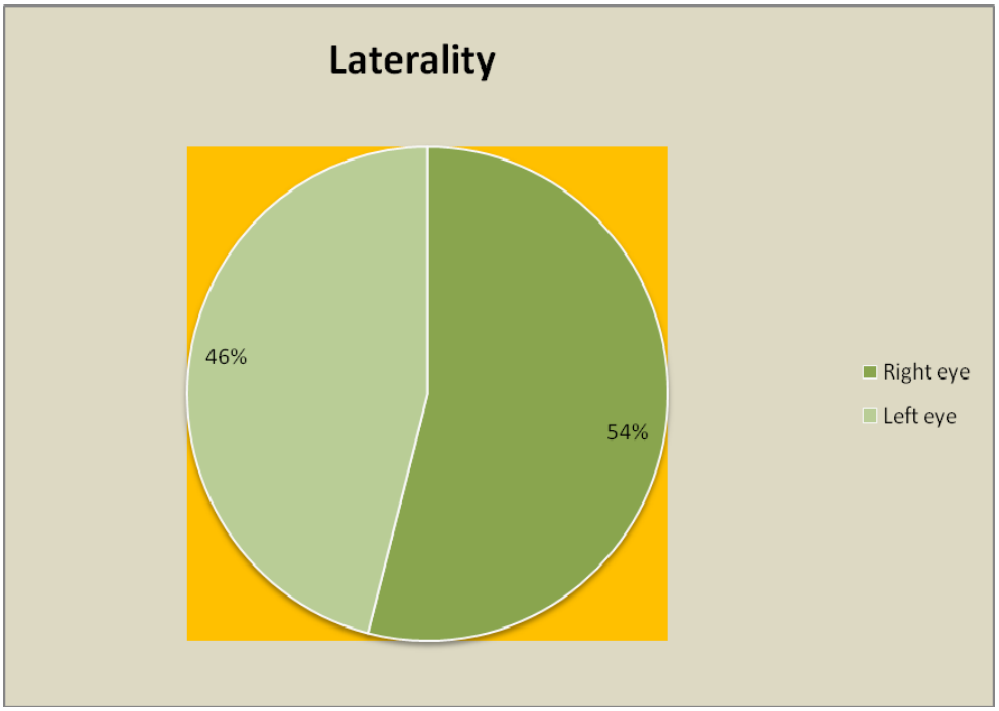
Out of 100 patients, 96 were male (96%), 4 were female (4%)



THE LATERALITY

Laterality	Right eye	Left eye
Frequency	56	44
Percentage	56%	44%

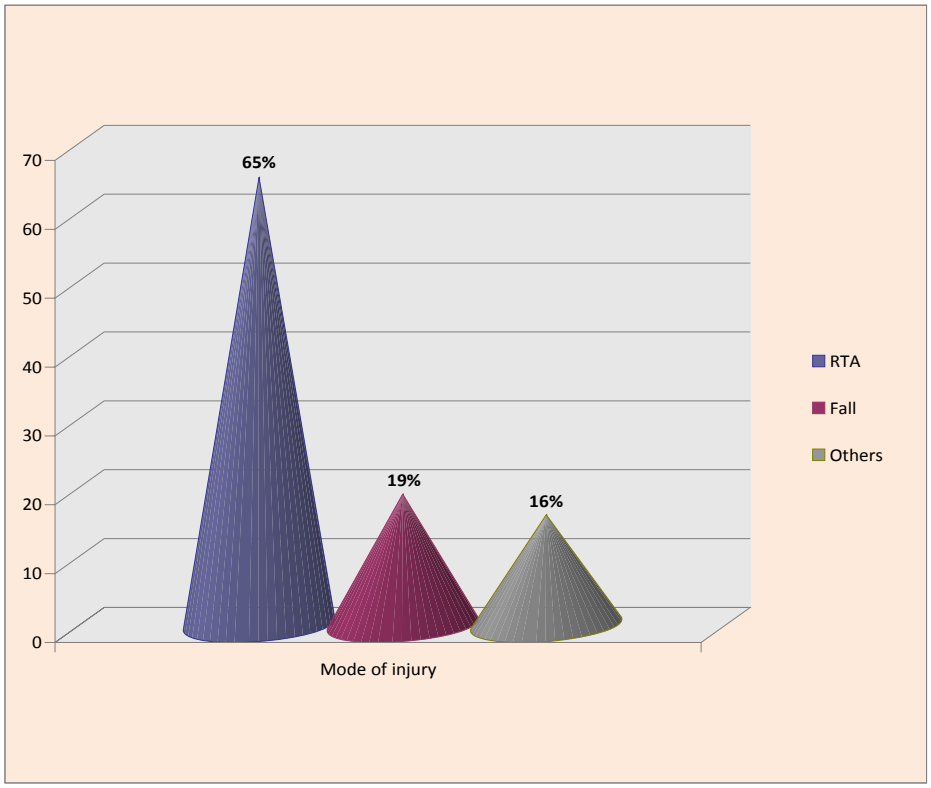
Of the 100 patients 56% of them had involvement of right eye and 44% of them had involvement of left eye.



MODE OF INJURY

Mode of injury	No of cases	Percentage
RTA	65	65%
Fall	19	19%
Others	16	16%

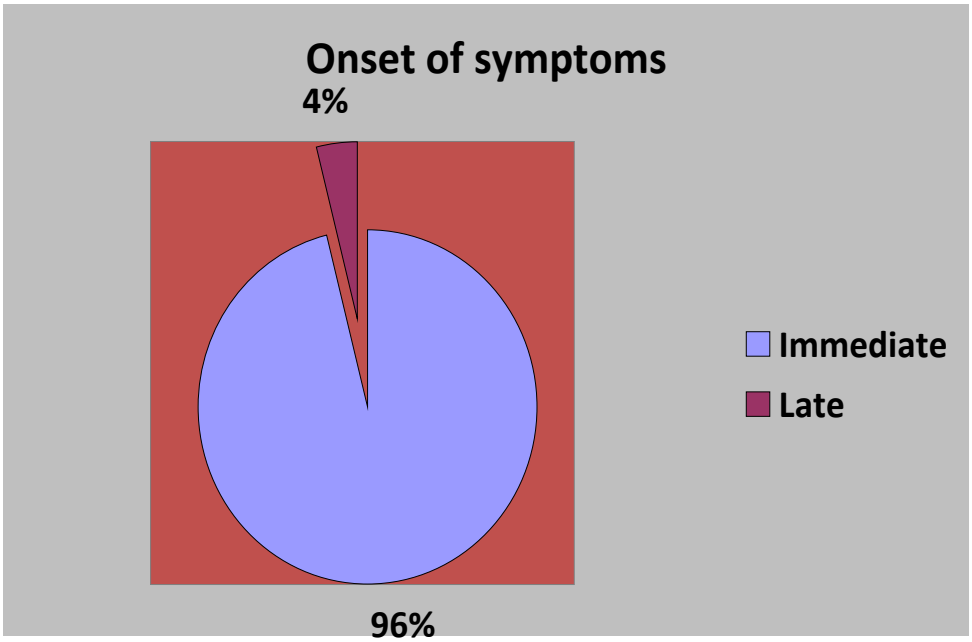
Motor vehicle and bicycle accidents for the majority of cases (65%) followed by fall (19%) and others.



ONSET OF SYMPTOMS

Immediate < 1 week	96%
Late > 1 week	4%

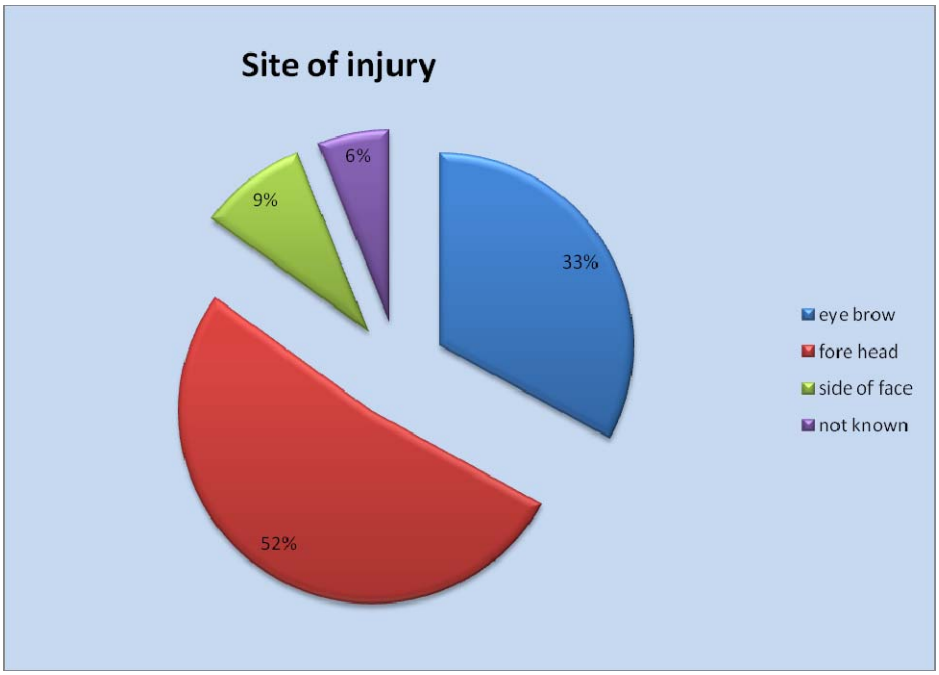
Majority of cases are immediate onset of visual symptoms (96%). Only 4% of cases presented with late onset of visual symptoms, this is because they would have developed the visual symptoms early but might have only noticed it later.



SITE OF INJURY

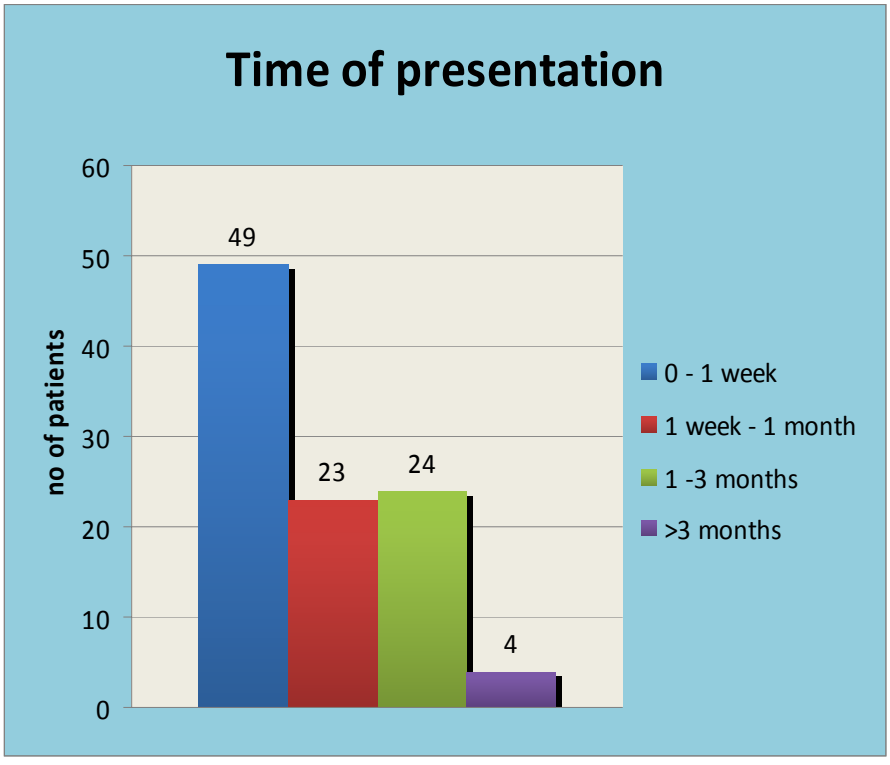
site of injury	eye brow	fore head	side of face	not known
no of cases	33	52	9	6
percentage	33%	52%	9%	6%

Fore head was the common site of injury (52%) followed by eye brow (33%) and side of face (9%).



TIME OF PRESENTATION

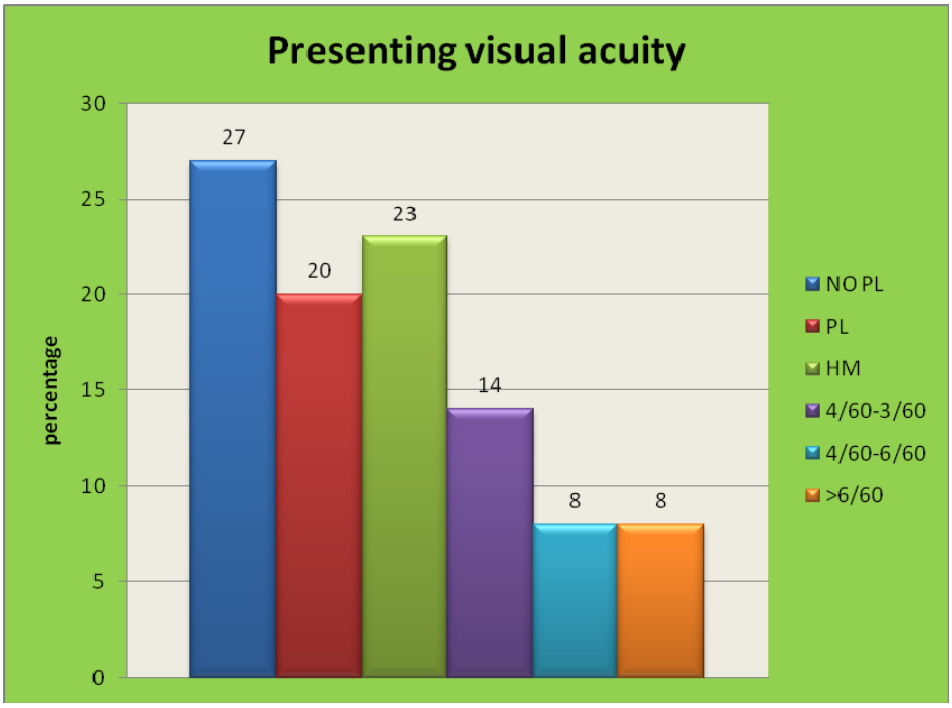
TIME OF PRESENTATION	NO OF CASES	PERCENTAGE
0 – 1 week	49	49%
1 week to 1 month	23	23%
1 – 3 months	24	24%
>3 months	4	4%



PRESENTING VISUAL ACUITY

VISUAL ACUITY	FREQUENCY	PERCENTAGE
NO PL	27	27%
PL+	20	20%
HM - CFCF	23	23%
1/60 – 3/60	14	14%
4/60 -6/60	8	8%
>6/60-6/18	8	8%

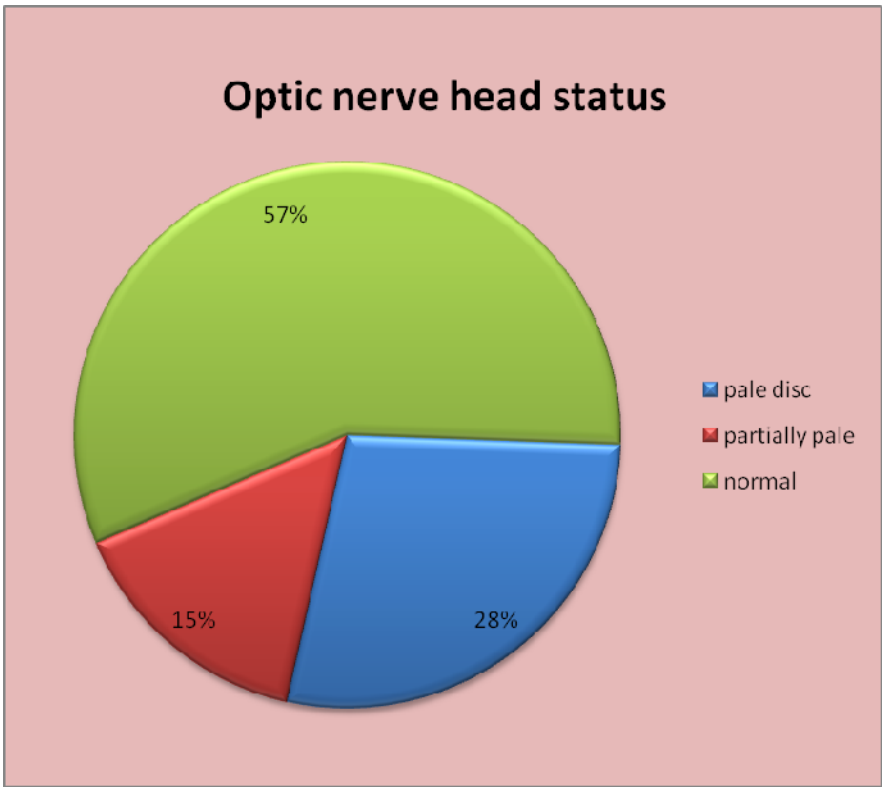
About 27% of patients had no light perception to light perception at the time of presentation. However 8% of patients had better than 6/60 at presentation.



OPTIC NERVE HEAD STATUS

Fundus	Frequency	Percentage
Normal	57	57%
Pale disc	28	28%
Partially pale	15	15%

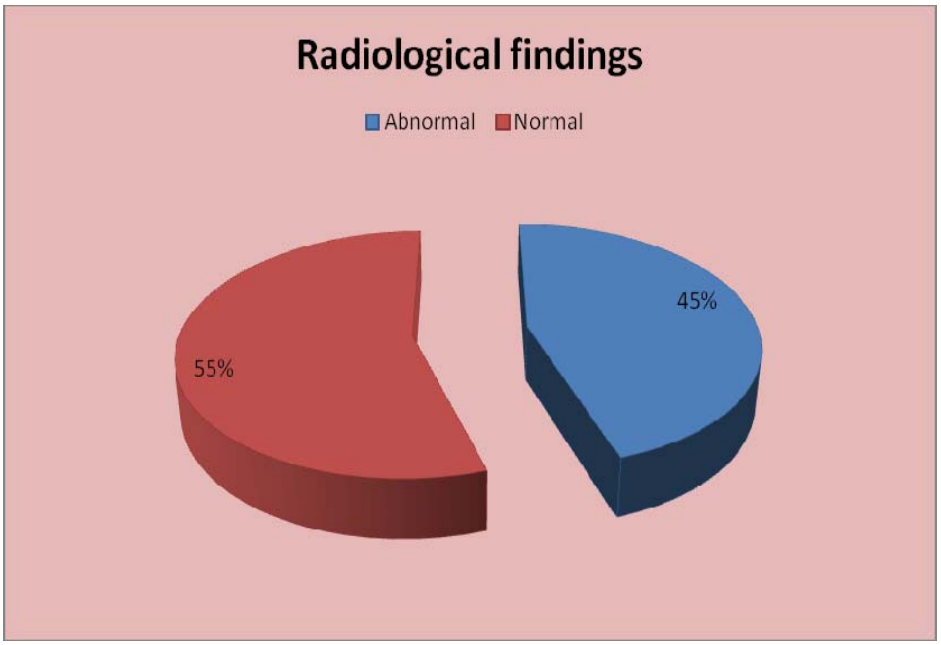
57% of patients had normal disc. 28% of patients had full pallor of disc and about 15% had partial pallor at the time of presentation.



RADIOLOGICAL FINDINGS

	No of Patients	Percentage
Abnormal	45	45%
Normal	55	55%

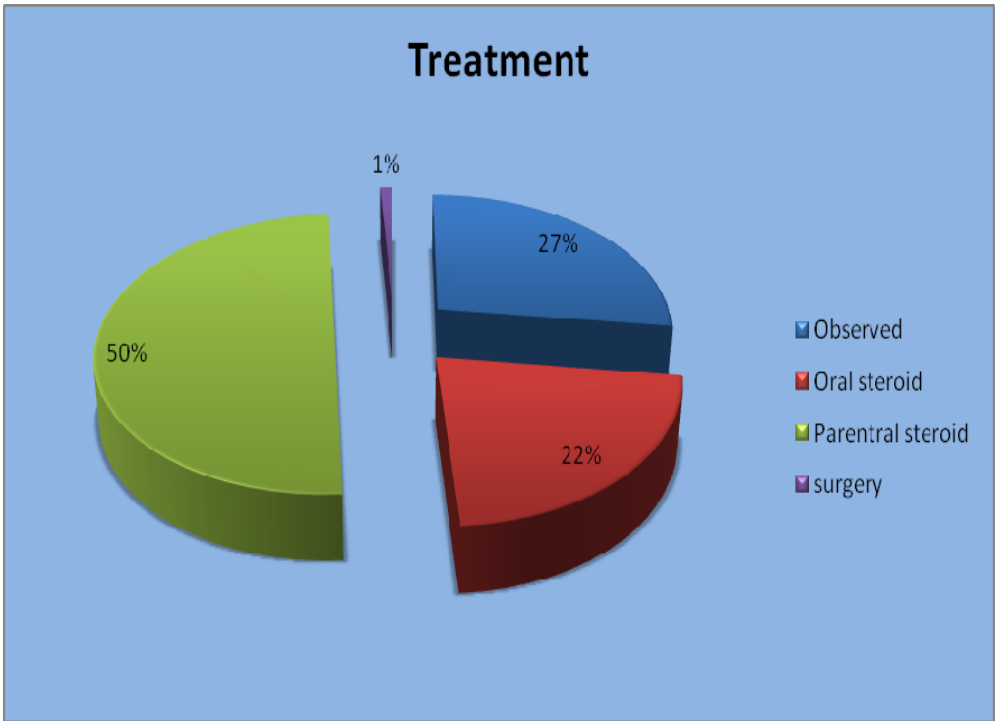
About 55% of cases presented without any radiological abnormality or fracture.45% had associated orbital, cranial or facial bone fractures evident radiologically.



TREATMENT

	Frequency	percentage
observed	27	27%
Oral steroids	22	22%
Parenteral steroids	50	50%
surgery	1	1%

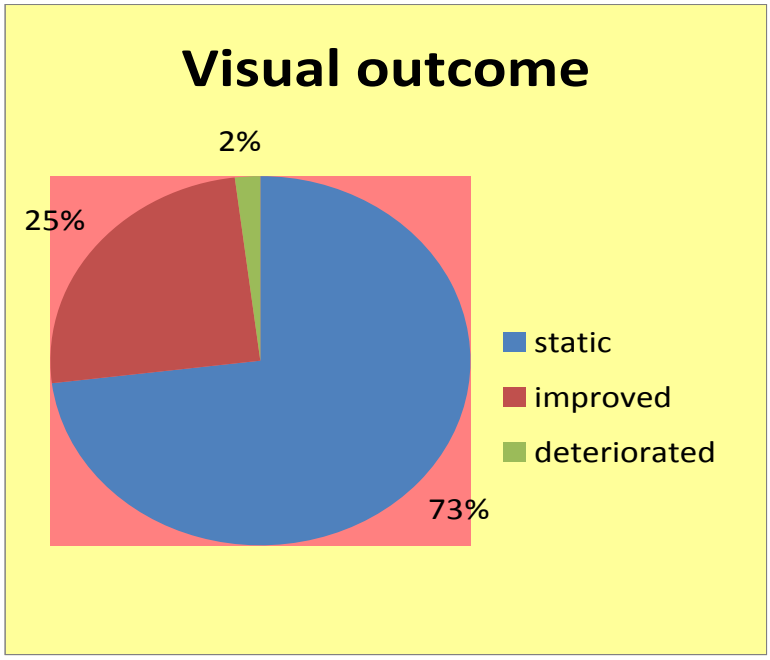
In 100 cases 27 cases were observed 22 cases were treated with oral steroids, 50 cases were given parenteral steroids followed by oral steroids. One case treated with endoscopic optic nerve decompression.



VISUAL OUTCOME

	No. of Patients	Percentages
Static	73	73%
Improved	25	25%
Deteriorated	2	2%

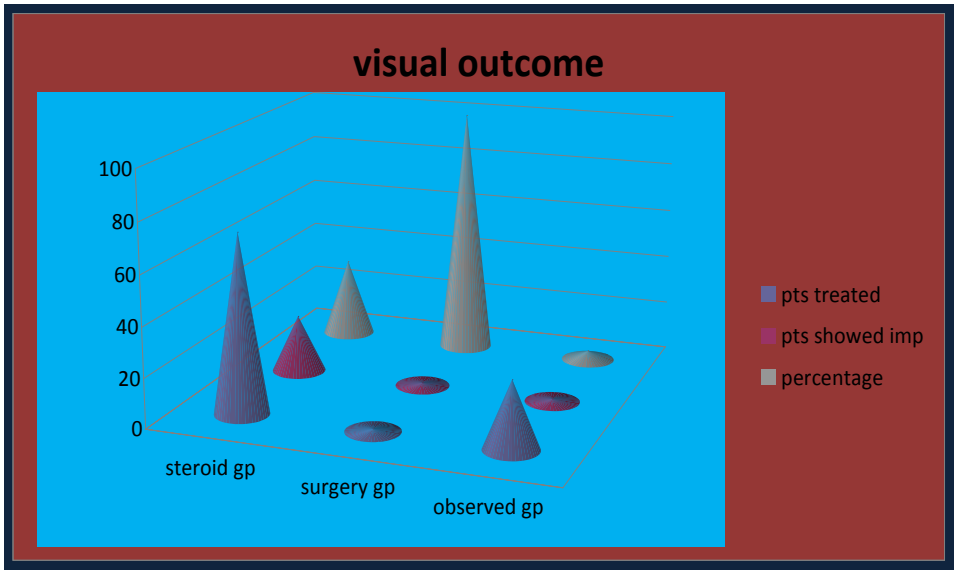
In the 100 cases only 25 cases showed improvement in visual function (three lines increase in VA in Snellen VA chart), 73 cases had static (patients with no improvement [69] and those with < three lines improvement [4]) and 2 cases had deteriorated visual function, both had poor VA at the time of presentation.



VISUAL OUTCOME WITH STEROID THERAPY

	No of pts treated	No of pts showed improved VA	Percentage
Steroid	72	23	31.94%
surgery	1	1	100%
Observed	27	1	3.70%

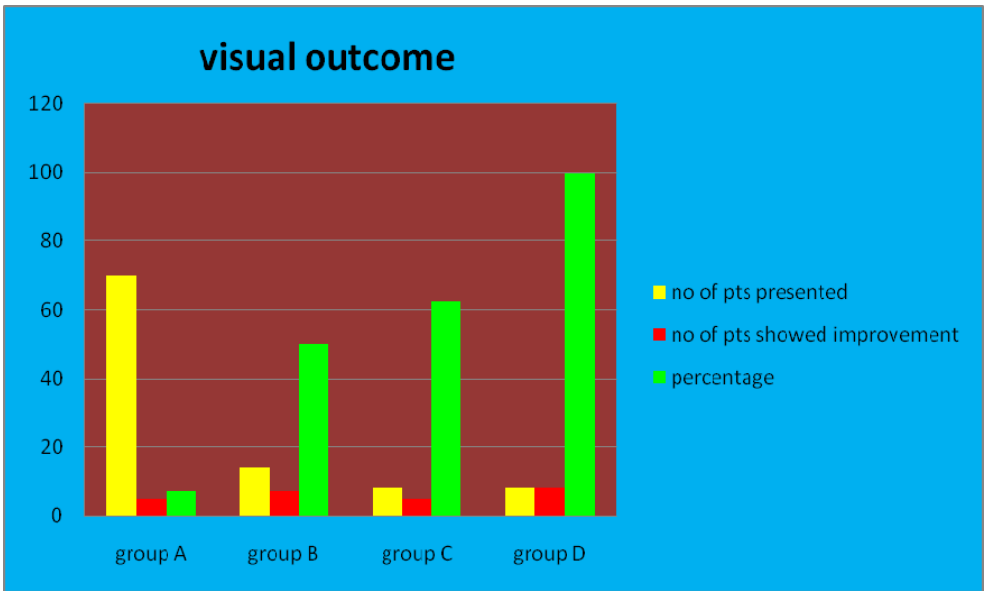
Out of 72 patients treated with corticosteroid (oral-22, parentral-50) only 23 patients (31.94%) showed visual improvement (oral-3, parentral-20). One patient who presented 1 month after injury had fracture medial wall of optic canal treated with endo nasal optic nerve decompression showed visual improvement from 4/60 to 6/36.



VISUAL IMPROVEMENT DEPENDING ON PRESENTING VISUAL ACUITY

	NO OF PTS PRESENTED	NO OF PTS SHOWED IMP	PERCENTAGE
No PL to HM	70	5	7.14%
1/60-3/60	14	7	50%
4/60-6/60	8	5	62.50%
6/36-6/18	8	8	100%

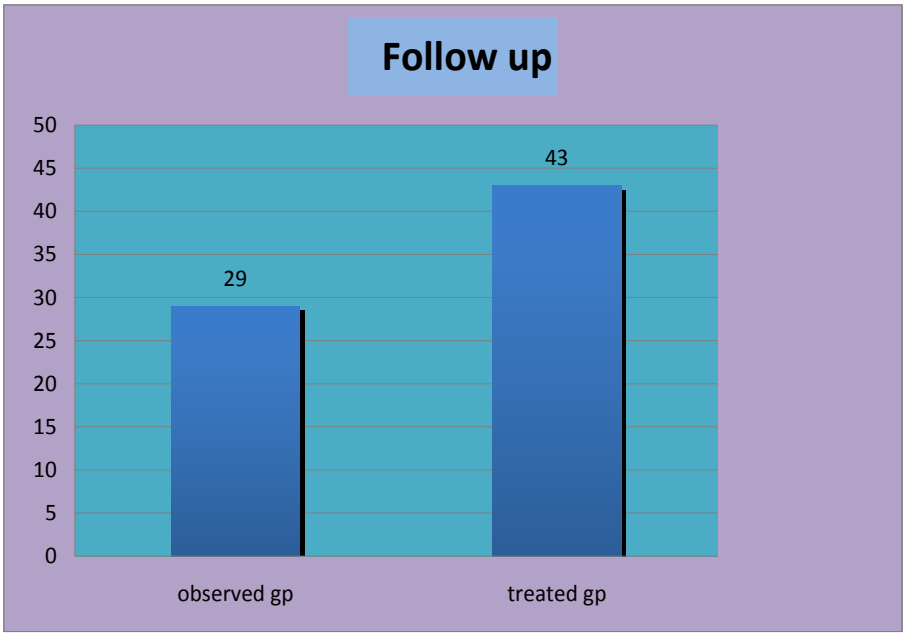
Among 100 patients 8 patients had VA > 6/60 and all showed improved visual function with treatment.



FOLLOW UP

	Percentage
Observed group	29%
Treated group	43%

Only 43% of treated group and 29% of observed group had come for follow up examination.



DISCUSSION

Hippocrates might have been the first to identify the phenomenon of acute and delayed vision loss after injuries placed to and slightly above the brow. While there is little controversy on the macroscopic mechanism of trauma theory, multiple hypothesis have been proposed at microscopic level of damage to the optic nerve including contusion necrosis, nerve fibre tears and nerve infarction secondary to closed space edema, haemorrhage, thrombosis, vasospasm, impingement by bone spicules and shearing of dural vessels in the optic canal.

In our study it is evident that traumatic optic neuropathy is devastating cause of permanent visual loss. Concussive force to head especially forehead transmits shock wave to optic canal. Visual loss is usually instantaneous.

Blunt trauma, penetrating injuries and self mutilation are the most common causes of optic nerve injury. Blunt trauma classically occurs following rapid deceleration injuries to the anterofrontal region of the head. Trauma to the outer third of the supraorbital rim is transmitted directly to the optic canal, where the optic nerve is tethered at both ends by dura. Conversely, the optic nerve is not taut in the orbit and protected by orbital fat and resistant to injury at this site.

The severity of the trauma does not always correlate with the visual loss. Incidents such as minor fall after tripping or hitting the side of the head against a solid object resulting in a frontal blow are adequate to produce a posterior traumatic optic neuropathy. The most common mechanism of injury is motor cycle accidents. Most of them are solo spills. Also the presence of or severity of the orbital fractures neither directly predicts the severity of visual loss nor determines the prognosis. Fracture of the medial orbital wall, floor, zygoma or optic canal may be present. One patient of optic canal fracture may regain normal vision without intervention but another with no fracture may present with no light perception that persists despite all intervention. Traumatic optic neuropathy is most often seen in young males in their 2^{st} or 3^{nd} decades of life. In one study, 5 patients older than 40 years of age were found to have a worse visual outcome independent of the mechanism of injury, severity of the visual loss or the interventions utilized.

Visual deficits range from mild decrease in visual acuity with subtle field defect to complete loss of light perception. In most cases, the visual loss is severe and instantaneous. Even seemingly trivial trauma may result in dramatic optic nerve impairment. The severity of the visual loss does not necessarily correlate with the degree of overall trauma.

Visual evoked potentials (Flash) have limited utility and may give false negative results prior to the onset of optic neuropathy. Although most patients with traumatic optic neuropathy have normal imaging studies, CT without contrast should be performed in all cases. Spiral CT allows rapid data acquisition in uncooperative adults and children. Even though fundus examination will reveal the following, imaging will allow identification of associated fractures, optic nerve avulsion, transection, optic nerve sheath hematoma and optic nerve compression due to an orbital hematoma. The optic nerve injury may not be isolated. Associated fractures and injuries can be identified when the scope of evaluation is expanded to include otolaryngology, oral, maxillofacial and neurosurgery colleagues. Magnetic resonance imaging is only indicated if intracranial injuries are present that are inadequately detailed with CT imaging. However concurrent orbito-facial fractures, extraocular nerve palsies and type of injury have no relevance to the final visual results.

This current study was carried over a period of 2 years, consecutive 100 patients were selected to determine the pattern of disease in a tertiary care centre like our institute. According to a study by Tang[14] (1986), out of 11 patients 36% showed visual improvement following maga dose steroid. In our study, 31.94% of the patients treated with steroids showed visual improvement. Analysing the demographic details in our study it is seen that most of our patients

(70%) were 20-40 years of age. Male patients were predominant in the study. This correlates with the higher rate of motor cycle accidents as the etiology. TON is a significant cause of post traumatic visual loss. The responsible blunt trauma to the frontal region may be minor or severe and accompanied by multiple adjacent fractures. Careful documentation of visual acuity, pupillary function and red desaturation is essential to guide management. CT imaging should be performed to document such abnormalities such as optic nerve avulsion, optic nerve sheath hematoma, orbital hematoma and optic canal fracture with fragments. If a structural abnormality is present or if the patient's visual acuity deteriorates on steroids, optic nerve decompression should be offered. Management of this disorder remains very controversial. In summary, a short course of high-dose steroid may be considered unless there is clear evidence of optic nerve transection or avulsion by clinical or radiographic criteria.

CONCLUSION

In our study the clinical profile of 100 eligible patients who presented with loss of vision following trauma during June 2008 – May 2010 were analyzed.

1. The age distribution of the patients were such that maximum no. of patients (70%) were in the age group of 20-40 years.

2. Males contributed 96 % while females contributed 4 %

3. The major etiology in our study was road traffic accident (65%) followed by falls (19%) and then assaults or other modes of injury (16%)

4. Most of the patients presented with Grade A visual loss. i.e. no PL to HM

5. In most cases development of visual symptoms after trauma was instantaneous.

6. Trauma around the eyebrow or frontal region was the commonest site of injury.

7. Most of the cases presented within 1 month from the onset of symptoms after trauma.

8. In most cases, optic disc was normal (57%) although some (28 %) had pale optic disc at presentation.

9. Of the 100 patients only 25% of them showed visual recovery to some degree with or without treatment.

10. Nearly 73% of them were left with permanent loss of vision.

11. About 2% of patients showed deteriorating visual outcome despite treatment.

12. Out of 72 patients treated with corticosteroid, only 23 patients had shown visual improvement (31.94%).

13. All the patients who presented with grade D (6/36 – 6/18) visual acuity showed visual improvement.

Considering the overall picture, it is evident that TON takes important place in permanent loss of vision in young males. Current clinical evidence is not sufficient to suggest the treatment option. Future developments in this field require randomised controlled clinical trials with extensive follow up to decide the treatment option. However it is evident that RTA especially two wheelers are at a particular risk and the incidence can be reduced by promoting education of riders to wear helmets.

PREVENTION IS BETTER THAN CURE

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PROFORMA

Analytical study of 100 cases of traumatic optic neuropathy Study No.

Name:

Op / Ip No:

Age:

Gender: [ ] 1. Male / 2. Female

Date of examination:

Laterality: [ ] 1. RE / 2. LE

Mode of injury: [ ] 1. RTA / 2. Fall / 3. Others

If others, specify:

Onset of symptoms: [ ]

Time of presentation: [ ]

Presenting VA: BCVA:

Site of injury: [ ] 1. Eye brow

2. Forehead

3. Side of face

4. Other parts of skull

Pupillary reaction: ☐ 1. Normal

2. RAPD

Fundus status: ☐ 1. Normal

2. Abnormal

If abnormal, specify:

Associated EOM Palsies: ☐ 1. Present

2. Not Present

Colour Vision: ☐ 1. Normal

2. Defective

Field defects: ☐ 1. Not present

2. Present

If present, specify:

Treatment: 1.Observed

2. Oral steroids

3. Parenteral steroids

4. Surgery

Follow Up Examination:

1st follow up:

Date of follow up:

VA at follow up: BCVA:

Status of VA: ☐ 1. Static

2. Improved

3. Deteriorated

Pupillary reaction: ☐ 1. Normal

2. RAPD

Fundus status: ☐ 1. Normal

2. Abnormal

If abnormal, specify:

Colour Vision: ☐ 1. Normal

2. Defective

Field defects: ☐ 1. Not present

2. Present

If present, specify:

2nd Follow up:

Date of follow up:

VA at follow up: BCVA:

Status of VA: ☐ 1. Static

2. Improved

3. Deteriorated

Pupillary reaction: ☐ 1. Normal

2. RAPD

Fundus status: ☐ 1. Normal

2. Abnormal

If abnormal, specify:

Colour Vision: ☐ 1. Normal

2. Defective

Field defects: ☐ 1. Not present

2. Present

If present, specify:

3rd follow up:

Date of follow up:

VA at follow up: BCVA:

Status of VA: ☐ 1. Static

2. Improved

3. Deteriorated

Pupillary reaction: ☐ 1. Normal

2. RAPD

Fundus status: ☐ 1. Normal

2. Abnormal

If abnormal, specify:

Colour Vision: ☐ 1. Normal

2. Defective

Field defects: ☐ 1. Not present

2. Present

If present, specify:

4th follow up:

Date of follow up:

VA at follow up: BCVA:

Status of VA: ☐ 1. Static

2. Improved

3. Deteriorated

Pupillary reaction: ☐ 1. Normal

2. RAPD

Fundus status: ☐ 1. Normal

2. Abnormal

If abnormal, specify:

Colour Vision: ☐ 1. Normal

2. Defective

Field defects: ☐ 1. Not present

2. Present

If present, specify:

KEY TO MASTER CHART

ALT	ALTITUDINAL FIELD DEFECT
BCVA	BEST CORRECTED VISUAL ACUITY
CC	CENTROCAECAL SCOTOMA
CFCF	COUNTING FINGER CLOSE TO FACE
EB	EYEBROW
EBS	ENLARGEMENT OF BLIND SPOT
EOM	EXTRA OCULAR MUSCLE PALSIES
FH	FORE HEAD
HM	HAND MOVEMENT
IP	IN PATIENT
LE	LEFT EYE
MOI	MODE OF INJURY
N	NORMAL
NI	NO IMPROVEMENT
NP	NOT POSSIBLE
OBS	OBSERVATION
OND	OPTIC NERVE DECOMPRESSION

OP	OUT PATIENT
OPS	OTHER PART OF SKULL
OS	ORAL STEROID
PCS	PARA CENTRAL SCOTOMA
PL	PERCEPTION OF LIGHT
PRE	PRESENT
PS	PARENTRAL STEROID
RAPD	RELATIVE AFFERENT PUPILLARY DEFECT
RE	RIGHT EYE
RTA	ROAD TRAFIC ACCIDENT
SF	SIDE OF FACE
TF	TUBULAR FIELD
UTQ	UPPER TEMPORAL QUADRANTANOPIA
VA	VISUAL ACUITY

LIST OF SURGERIES

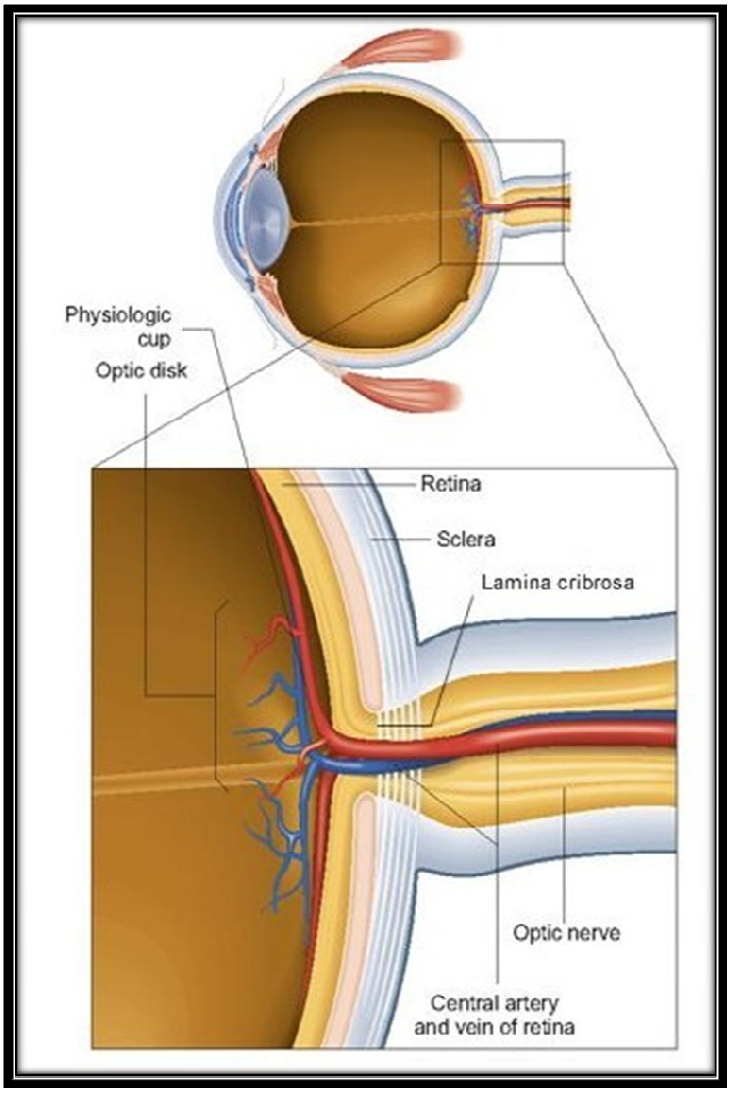
S.No	Name	Age/ sex	IP No	Diagnosis	Procedure
1.	Vasantha	50/F	430987	RE MC/LE Aphakia	RE ECCE
2.	Jinna	65/M	431090	BE IMC	LEECCE/PCIOL
3.	Jabastin	59/M	425152	RE PCIOL/LE MC	LE ECCE/PCIOL
4.	Indurani	45/F	435162	RE IMC/LE MC	LE ECCE/PCIOL
5.	Kasthuri	60/F	436477	BC IMC	LE SICS/PCIOL
6.	Thatchayini	40/F	436693	RE MC/LE IMC	RE ECCE/PCIOL
7.	Eswari	50/F	436946	RE MC/LE IMC	RE ECCE/PCIOL
8.	Shanmugam	65/M	437265	BE IMC	RE SICS/PCIOL
9.	Subbulakshmi	55/F	437251	BE NC	RE SICS/PCIOL
10	Thara	62/F	437555	BE NS LE>RE	LE SICS/PCIOL
11	Gowri	55/F	437681	RE IMC/LE PCIOL	RE SICS/PCIOL
12	Backiam	45/F	437768	LE Phacolyticglaucoma	LE SICS/PCIOL
13	Rajamannar	65/M	437556	BE PSCC	LE SICS/PCIOL
14	Chokkalingam	54/M	437994	RE PCIOL/LE MC	LE SICS/PCIOL
15	Kamsala	55/F	438261	RE MC/LE PCIOL	RE SICS/PCIOL

S.No	Name	Age/ sex	IP No	Diagnosis	Procedure
16	Soodamani	70/F	439012	BE IMC RE>LE	LE SICS/PCIOL
17	Kamarbanu	64/F	439118	RE PCIOL/LE IMC	LE Phaco/PCIOL
18	Devaraj	64/M	439234	BE IMC	LE Phaco/PCIOL
19	Lakshmi	60/F	51267	LE Pterygium	Excision with autograft
20	Shankar	22/M	53142	RE Chalazion	Incision & curettage
21	Kalyani	50/F	53763	RE Pterygium	Excision with AMG
22	Meenatchi	55/F	438654	RE Post traumatic endophthalmitis	Intravitreal amikacin
23	Rajendren	45/M	439387	LE Non healing fungal corneal ulcer	LE Theraputic keratoplasty
24	Lalitha	54/F	438769	RE fungal corneal ulcer	RE AC wash with Amphotericin B
25	Rajesh	60/M	56375	RE chronic dacryocystitis	RE Dacryocystectomy
26	Gejalakshmi	38/F	439432	RE Facial palsy	RE Lateral Tarsorrhaphy
27	Ramani	66/F	435267	RE Upper lid abscess	Incision & drainage
28	Saradha	22/F	439765	LE post traumatic panophthalmitis	LE Evisceration PMMA implant
29	Narayanan	58/M	439897	LE Secondary glaucoma	LE trabeculectomy
30	Ramya	23/F	439989	LE Sensory exotropia	LE LR recession MR resection

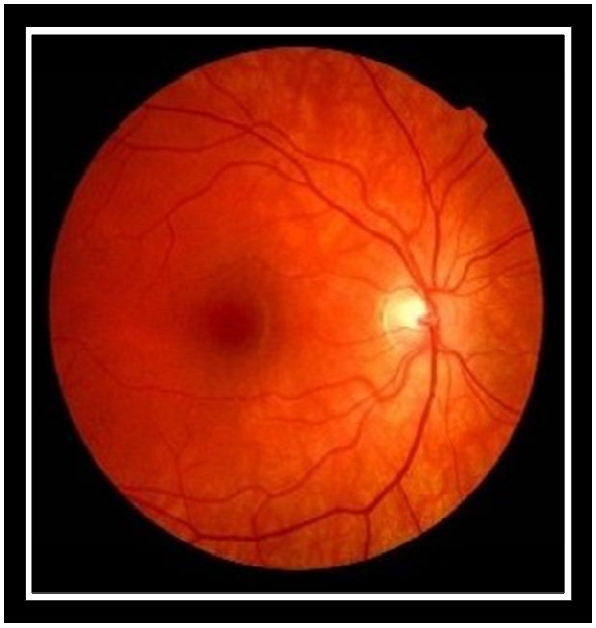
Sl. NO.	NAME	IP NO/ OP NO.	AGE	Sex	DATE	LATERALITY	MOI	ONSET	TIME-PRE	VA AT PRE	BC VA	SITE-INJURY	EOM	PUPILLLRY REACTION	FUNDUS	COLOR VISION	Type of Field	RADIOIOGICAL FINDING	TREATMENT
1	Anand	425412	27	M	13.6.08	RE	RTA	1	1	HM+	NI	EB	2	RAPD	Normal	NP	NP	#lat wall of orbit, #zygoma, #maxilla, #frontal	PS
2	Gunasekar	85855	21	M	21.6.08	LE	RTA	1	3	No PL	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
3	Shankar	86723	27	M	28.6.08	LE	assault	1	3	1/60.	NI	FH	2	RAPD	pale disc	RAPD	NP	N	OBS
4	Baskar	87434	43	M	4.7.08	RE	RTA	1	2	CFCF	NI	FH	2	RAPD	Normal	NP	NP	N	OS
5	Munuswamy	88256	30	M	16.7.08	RE	RTA	1	3	No PL	NI	FH	2	RAPD	pale disc	NP	NP	#floor of orbit	OBS
6	Kajendren	425526	44	M	22.7.08	LE	Fall	1	3	No PL	NI	FH	2	RAPD	Normal	NP	NP	N	OBS
7	Siva	426114	25	M	30.7.08	RE	RTA	1	1	HM+	NI	EB	2	RAPD	Normal	NP	NP	N	PS
8	Srinivasan	426664	14	M	1.8.08	RE	Fall	1	3	No PL	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
9	Nagendren	429760	28	M	12.8.08	LE	Fall	2	1	No PL	NI	EB	2	RAPD	Normal	NP	NP	#medial wall orbit	PS
10	Rajesh	430103	35	M	17.8.08	LE	RTA	1	3	CFCF	NI	SF	2	RAPD	pale disc	NP	NP	N	OBS
11	Ravi	88948	30	M	12.09.08	RE	RTA	1	2	No PL	NI	EB	2	RAPD	pale disc	NP	NP	N	OS
12	Ethiraj	89309	30	M	14.9.08	LE	RTA	1	4	1/60.	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
13	Mani	89648	30	M	19.9.08	RE	Fall	1	3	No PL	NI	EB	2	RAPD	temporal pallor	NP	NP	N	OBS
14	Kathirvel	433856	36	M	29.9.08	RE	RTA	1	1	6/60.	6/36.	OPS	2	RAPD	Normal	RAPD	CC	#lat wall of rt. orbit , #zygoma	PS
15	Parasuraman	433923	33	M	3.10.08	RE	RTA	1	3	PL +	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
16	Anbu	433345	16	M	13.10.08	RE	RTA	1	1	PL +	NI	FH	2	RAPD	temporal pallor	NP	NP	#maxilla	PS
17	Azhagesan	433521	38	M	19.10.08	LE	RTA	1	3	5/60.	6/18.	FH	2	RAPD	temporal pallor	RAPD	TF	N	OBS
18	Iyyappan	89876	36	M	24.10.08	RE	injury with ball	1	4	CFCF	NI	EB	2	RAPD	temporal pallor	NP	NP	N	OBS
19	Babu	434274	38	M	2.11.08	RE	Fall	1	1	1/60.	6/24.	FH	2	RAPD	Normal	RAPD	N	N	PS
20	Ranjithkumar	89930	17	M	5.11.08	RE	RTA	1	4	No PL	NI	OPS	2	RAPD	temporal pallor	NP	NP	N	OBS
21	Thanikachalam	434328	49	M	6.11.08	RE	Fall	1	1	HM+	6/18.	SF	2	RAPD	Normal	RAPD	TF	N	PS
22	Mujibullah	434822	23	M	16.11.08	RE	RTA	1	1	PL +	1/60.	FH	2	RAPD	Normal	NP	NP	N	PS
23	Baskar	90172	30	M	4.12.08	LE	assault	1	1	6/24.	6/9P	SF	2	RAPD	Normal	RAPD	N	#lateral wall of orbit	PS
24	Krishnan	436158	42	M	7.12.08	RE	RTA	2	2	HM+	NI	EB	2	RAPD	Normal	NP	NP	N	OS
25	Mahendren	439032	16	M	18.12.08	RE	RTA	1	1	6/60.	6/24.	FH	2	RAPD	Normal	RAPD	N	#sphenoid	PS
26	Selvam	90259	33	M	31.12.08	LE	RTA	1	3	No PL	NI	SF	2	RAPD	pale disc	NP	NP	#lat.wall	OBS
27	Kannan	439428	30	M	2.1.09	LE	RTA	1	1	No PL	NI	FH	2	RAPD	pale disc	NP	NP	#lat.wall	PS
28	Baskar	90365	30	M	14.1.09	RE	RTA	1	3	HM+	NI	EB	2	RAPD	pale disc	NP	NP	sphenoid, #frontal, #temporal	OBS
29	Murugan	439965	42	M	24.1.09	LE	assault	1	1	CFCF	NI	EB	2	RAPD	temporal pallor	NP	NP	#roof	PS
30	Gowri	443457	50	F	31.1.09	LE	hit on wall	1	1	CFCF	NI	FH	2	RAPD	Normal	NP	NP	N	PS
31	Selvaprakash	443800	25	M	1.2.09	RE	RTA	1	1	No PL	NI	FH	2	RAPD	Normal	NP	NP	bone	PS
32	Lakhmi	443885	35	F	6.2.09	LE	Fall	1	1	1/60.	6/9.	FH	2	RAPD	Normal	NP	NP	N	PS
33	Jeevanandham	445132	29	M	8.02.09	LE	RTA	1	2	PL +	1/60.	SF	2	RAPD	Normal	RAPD	ALT	#maxilla	PS
34	Mani	445157	40	M	27.2.09	RE	RTA	1	1	HM+	NI	EB	2	RAPD	Normal	NP	NP	# Rt lat.wall, # rt.zygoma	PS
35	Sivagnanam	445206	35	M	5.3.09	RE	RTA	2	1	PL +	6/24.	FH	2	RAPD	Normal	NP	NP	N	PS
36	Kuselan	445249	50	M	7.3.09	RE	RTA	1	2	PL +	NI	EB	2	RAPD	Normal	NP	NP	#greater wing,# rt zygoma, # maxilla	OS
37	Tamilvanan	445385	30	M	22.3.09	LE	Fall	1	1	6/18.	6/6.	FH	2	RAPD	Normal	RAPD	N	#medial, lateral, floor of orbit	PS
38	Moorthy	445770	39	M	30.3.09	LE	RTA	1	1	2/60.	6/18.	EB	2	RAPD	pale disc	RAPD	N	N	PS
39	Koteeswaran	445875	25	M	10.4.09	RE	RTA	1	1	No PL	HM+	FH	2	RAPD	Normal	NP	NP	#Frontal, temporal, greater wing sphenoid bone	PS

Sl. NO.	NAME	IP NO/ OP NO.	AGE	Sex	DATE	LATERALITY	MOI	ONSET	TIME-PRE	VA AT PRE	BC VA	SITE-INJURY	EOM	PUPILLRY REACTION	FUNDUS	COLOR VISION	Type of Field	RADIOIOGICAL FINDING	TREATMENT
40	Mohan	446453	32	M	10.4.09	RE	RTA	1	1	6/60.	6/6.	EB	2	RAPD	Normal	N	N	N	PS
41	Ramachandren	446478	27	M	24.4.09	LE	assault	1	1	No PL	NI	EB	2	RAPD	pale disc	NP	NP	#roof	PS
42	Muralidharan	90457	32	M	25.4.09	LE	RTA	1	3	PL +	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
43	Karthick	90514	21	M	3.5.09	RE	RTA	1	2	PL +	NI	EB	2	RAPD	Normal	NP	NP	N	OS
44	Sathish	449429	22	M	24.5.09	RE	RTA	1	2	6/36.	6/12.	OPS	2	RAPD	Normal	RAPD	PCS	#orbit inf & lat wall, # maxilla, # zygoma, # temporal bone	OS
45	Kannan	90589	26	M	28.5.09	RE	RTA	1	3	No PL	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
46	Bazheer ahmad	90598	39	M	30.5.09	LE	Fall	1	2	6/36.	6/9P	EB	2	RAPD	Normal	RAPD	N	#roof, #maxilla, #zygoma, #temporal bone	OS
47	Uvaraj	449473	14	M	1.6.09	LE	Fall	1	1	6/24.	6/6P	FH	2	RAPD	Normal	RAPD	ALT	N	PS
48	Umanandam	449891	14	M	6.6.09	LE	hit against wall	1	3	No PL	NI	EB	2	RAPD	pale disc	NP	NP	N	OBS
49	Mubarak	90624	26	M	15.6.09	RE	RTA	1	3	1/60.	NI	OPS	2	RAPD	pale disc	RAPD	NP	#lat.wall,#greater wing of sphenoid, #zygoma, #temporal bone	OBS
50	Chandren	449891	26	M	17.6.09	LE	RTA	1	2	3/60.	NI	FH	2	RAPD	Normal	RAPD	UTQ	N	OS
51	Mani	449900	43	M	18.6.09	LE	assault	1	2	6/18.	6/6P	EB	2	RAPD	Normal	RAPD	N	retrobulbar haemorrhage	OS
52	Ramesh	90772	30	M	8.7.09	RE	Fall	1	2	CFCF	NI	FH	2	RAPD	Normal	NP	NP	#lat wall	OS
53	Sivakumar	90870	32	M	14.7.09	RE	assault	2	3	PL +	NI	SF	2	RAPD	pale disc	NP	NP	N	OBS
54	Paramasivam	450989	37	M	19.7.09	LE	RTA	1	2	No PL	NI	FH	2	RAPD	temporal pallor	NP	NP	#frontal bone	OS
55	Anandhan	451100	42	M	22.7.09	LE	RTA	1	3	No PL	NI	EB	2	RAPD	pale disc	NP	NP	N	OBS
56	Tamilselvan	451478	22	M	26.7.09	LE	assault	1	1	2/60.	6/60.	FH	2	RAPD	Normal	RAPD	ALT	#medial, lateral, roof of orbit, #maxilla, #zygoma, #frontal bone	PS
57	Jagajeevanram	91489	45	M	26.7.09	LE	RTA	1	2	CFCF	NI	EB	2	RAPD	Normal	NP	NP	#frontal,#temporal,#maxilla,#roof,#sphenoid	OS
58	Durga	451552	17	F	6.8.09	RE	RTA	1	3	PL +	NI	FH	2	RAPD	pale disc	NP	NP	optic nerve edema	OS
59	Hemraj	451614	27	M	9.8.09	RE	RTA	1	1	HM+	NI	EB	2	RAPD	Normal	RAPD	NP	#frontal bone	PS
60	Chandren	451619	34	M	18.8.09	LE	RTA	1	2	PL +	NI	FH	2	RAPD	pale disc	NP	NP	N	OS
61	Krishnamoorthy	451927	33	M	18.8.09	RE	RTA	1	1	1/60.	NI	EB	2	RAPD	Normal	NP	NP	N	PS
62	Kannan	452476	15	M	16.9.09	RE	RTA	1	2	4/60.	6/36.	FH	2	RAPD	temporal pallor	RAPD	ALT	#medial wall of optic canal	OCD
63	Mohamadyasak	452506	17	M	17.9.09	LE	Fall	1	1	HM+	NI	FH	2	RAPD	pale disc	NP	NP	#greater wing, # roof, floor, lat wall of orbit	PS
64	Karthikeyan	452595	12	M	19.9.09	RE	RTA	1	1	No PL	NI	OPS	2	RAPD	Normal	NP	NP	N	PS
65	Krishnakumar	452624	29	M	8.10.09	RE	RTA	1	1	6/60.	NI	FH	2	RAPD	temporal pallor	RAPD	ALT	#lateral wall of orbit	PS
66	Vijayakumar	91967	26	M	29.10.09	LE	RTA	1	2	HM+	NI	FH	2	RAPD	temporal pallor	NP	NP	N	OS
67	Babu	452970	21	M	6.11.09	RE	RTA	1	1	PL +	NI	OPS	2	RAPD	Normal	NP	NP	N	PS
68	Raja	92123	26	M	8.11.09	LE	Fall	1	3	No PL	NI	FH	2	RAPD	pale disc	NP	NP	N	OBS
69	swamikkannu	453143	52	M	10.11.09	RE	RTA	1	1	1/60.	NI	FH	2	RAPD	Normal	RAPD	N	N	PS
70	Ravi	453195	43	M	2.12.09	RE	Fall	1	1	5/60.	6/24.	SF	2	RAPD	Normal	RAPD	ALT	N	PS
71	Rajaram	453221	18	M	14.12.09	LE	assault	1	1	PL +	NI	FH	2	RAPD	Normal	NP	NP	N	PS
72	Ashok	83467	29	M	23.12.09	RE	RTA	1	2	PL +	NI	EB	2	RAPD	temporal pallor	NP	NP	#lat wall	OS
73	Elango	83765	21	M	31.12.09	LE	RTA	1	2	HM+	NI	FH	2	RAPD	temporal pallor	NP	NP	#maxilla	OS

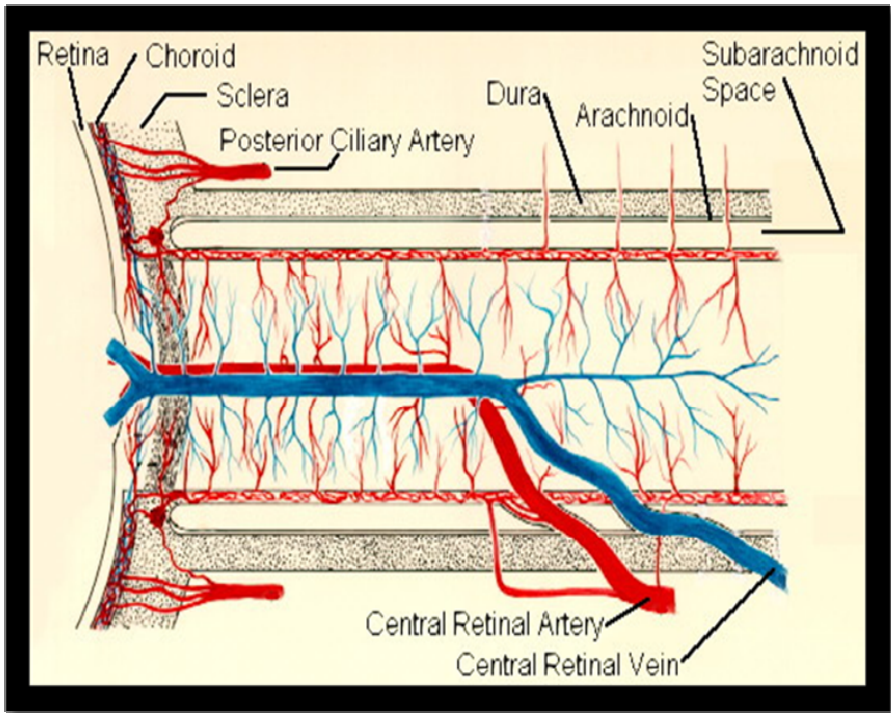
Sl. NO.	NAME	IP NO/ OP NO.	AGE	Sex	DATE	LATERALITY	MOI	ONSET	TIME-PRE	VA AT PRE	BC VA	SITE-INJURY	EOM	PUPILLLRY REACTION	FUNDUS	COLOR VISION	Type of Field	RADIOIOGICAL FINDING	TREATMENT
74	Sakthi	83965	20	M	1.1.10	LE	RTA	1	2	6/60.	6/36.	FH	2	RAPD	Normal	RAPD	ALT	N	OS
75	Sankar	453334	33	M	4.1.10	LE	Fall	1	1	PL +	NI	EB	2	RAPD	Normal	NP	NP	#roof	PS
76	Veeraiyan	84387	47	M	14.1.10	RE	RTA	1	4	No PL	NI	FH	2	RAPD	pale disc	NP	NP	#frontal bone	OBS
77	Nirmal	84465	19	M	16.1.10	RE	RTA	1	2	HM+	NI	EB	2	RAPD	temporal pallor	NP	NP	#lat wall, #zygoma, #maxilla	OS
78	Nagaraj	453387	24	M	26.1.10	LE	RTA	1	1	2/60.	6/60.	FH	2	RAPD	Normal	RAPD	ALT	N	PS
79	Ulaganathan	453423	31	M	3.2.10	LE	Fall	1	1	HM+	NI	FH	2	RAPD	Normal	NP	NP	#frontal	PS
80	Peter	84878	17	M	5.2.10	RE	RTA	1	3	No PL	NI	EB	2	RAPD	temporal pallor	NP	NP	N	OBS
81	Saradha	453496	28	F	16.2.10	RE	RTA	1	1	6/24.	6/9.	SF	2	RAPD	Normal	RAPD	N	N	PS
82	Saied raj	453489	25	M	24.2.10	LE	assault	1	1	1/60.	6/60.	FH	2	RAPD	Normal	RAPD	N	N	PS
83	David	453567	41	M	6.3.10	RE	RTA	1	1	PL +	NI	FH	2	RAPD	Normal	NP	NP	#temporal bone	PS
84	Govindhan	85321	36	M	7.3.10	LE	RTA	1	3	PL +	NI	EB	2	RAPD	pale disc	NP	NP	#lat wall	OBS
85	Kopinath	453589	27	M	15.3.10	RE	RTA	1	1	HM+	2/60.	FH	2	RAPD	Normal	NP	NP	N	PS
86	Ragavendren	85643	26	M	29.3.10	RE	assault	1	2	2/60.	NI	EB	2	RAPD	Normal	RAPD	EBS	N	OS
87	Mohan	453661	32	M	31.3.10	RE	RTA	1	1	6/36P	6/9.	FH	2	RAPD	Normal	RAPD	N	N	PS
88	Prabu	453693	24	M	31.3.10	LE	Fall	1	1	PL +	No PL	FH	2	RAPD	Normal	NP	NP	#maxilla, #lat wall	PS
89	Ramu	453699	28	M	1.4.10	RE	RTA	1	1	No PL	NI	EB	2	RAPD	Normal	NP	NP	#roof	PS
90	Bakiaraj	453702	38	M	9.4.10	RE	assault	1	1	PL +	NI	FH	2	RAPD	Normal	NP	NP	N	PS
91	Cidambaram	86232	49	M	10.4.10	RE	RTA	1	3	No PL	NI	EB	2	RAPD	pale disc	NP	NP	#zygoma	OBS
92	Ravichandren	453787	30	M	16.4.10	LE	Fall	1	1	HM+	No PL	FH	2	RAPD	Normal	NP	NP	N	PS
93	Thangamani	86456	21	M	16.4.10	RE	RTA	1	3	No PL	NI	EB	2	RAPD	pale disc	NP	NP	#lat wall, #zygoma, #maxilla	OBS
94	Prakash	453832	35	M	22.4.10	LE	RTA	1	1	No PL	NI	FH	2	RAPD	Normal	NP	NP	N	PS
95	Chellaiah	86675	29	M	28.4.10	RE	RTA	1	2	HM+	NI	FH	2	RAPD	Normal	NP	NP	N	OS
96	Annamalai	453879	45	M	2.5.10	RE	assault	1	1	No PL	NI	FH	2	RAPD	Normal	NP	NP	#temporal bone	PS
97	Premkumar	86878	32	M	5.5.10	RE	RTA	1	3	PL +	NI	EB	2	RAPD	pale disc	NP	NP	N	OBS
98	Yokesh	453892	26	M	12.5.10	LE	Fall	1	1	3/60.	6/36.	SF	2	RAPD	Normal	RAPD	N	N	PS
99	Saravanan	453956	35	M	17.5.10	LE	RTA	1	1	HM+	1/60.	FH	2	RAPD	temporal pallor	NP	NP	N	PS
100	Manikandan	86498	23	M	17.5.10	RE	wall	1	2	No PL	NI	EB	2	RAPD	pale disc	NP	NP	#lat wall haematoma	OS



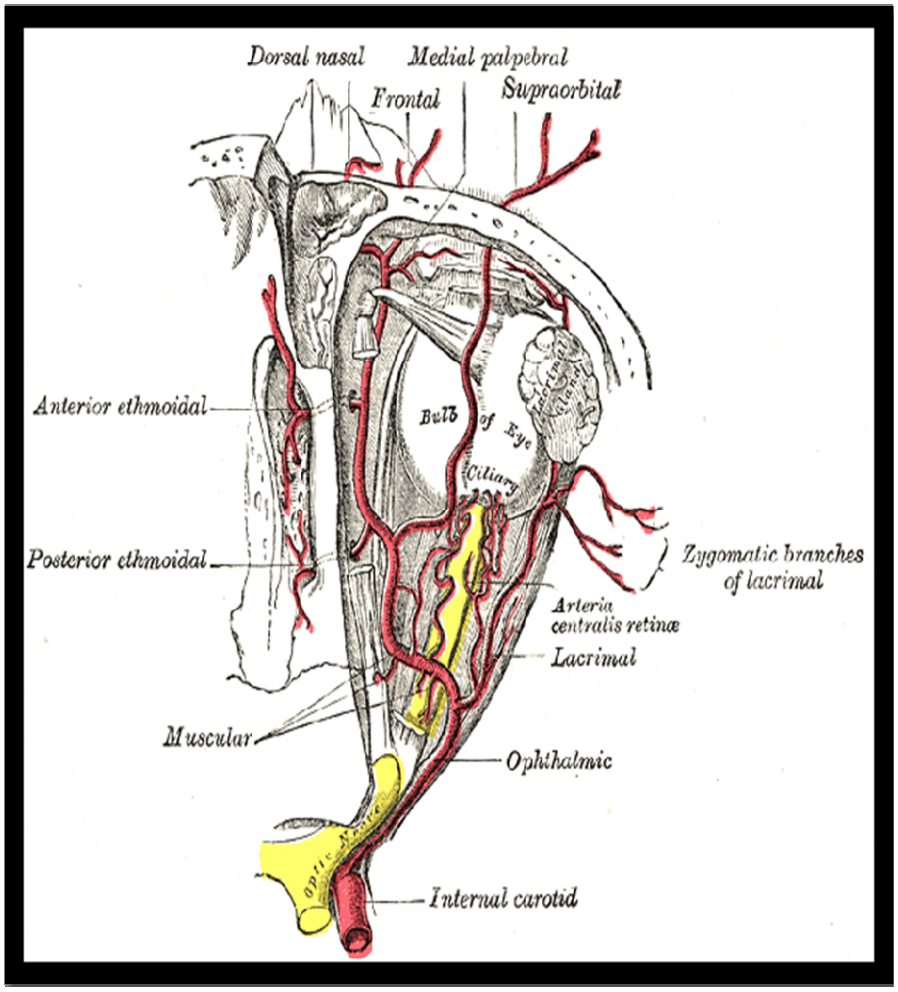
Intra ocular part of optic nerve



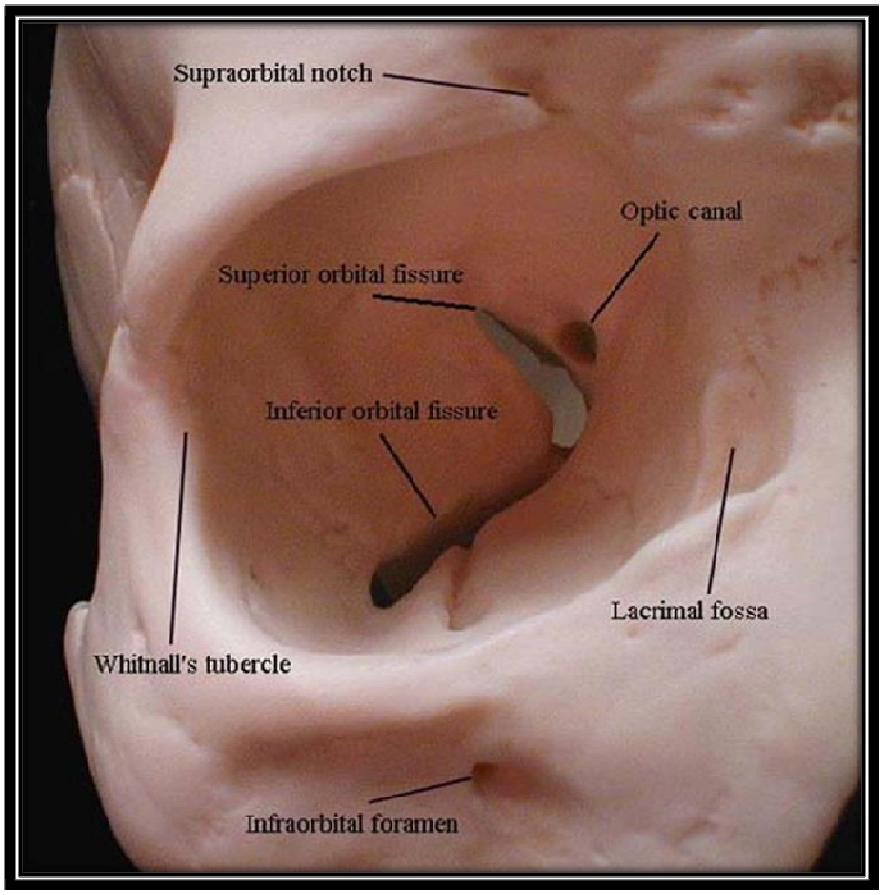
Fundus picture of normal optic nerve head



Blood supply of intraocular part and anterior portion of orbital part of optic nerve

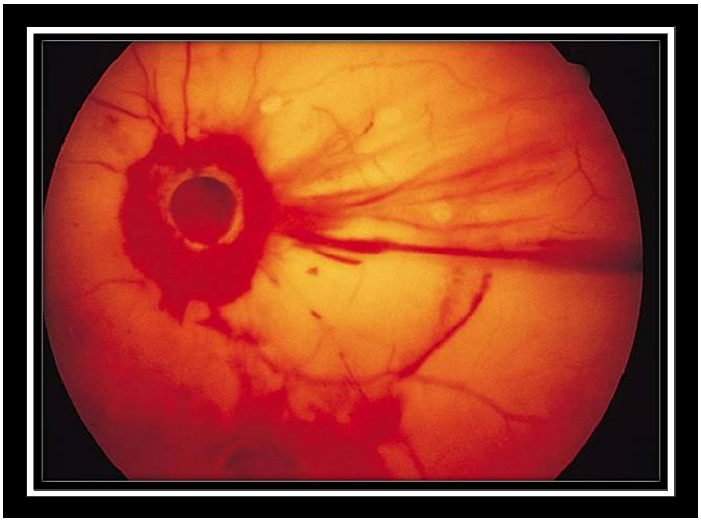


Blood Supply



Bony orbit showing the optic canal

Histology picture of perineural hematoma.

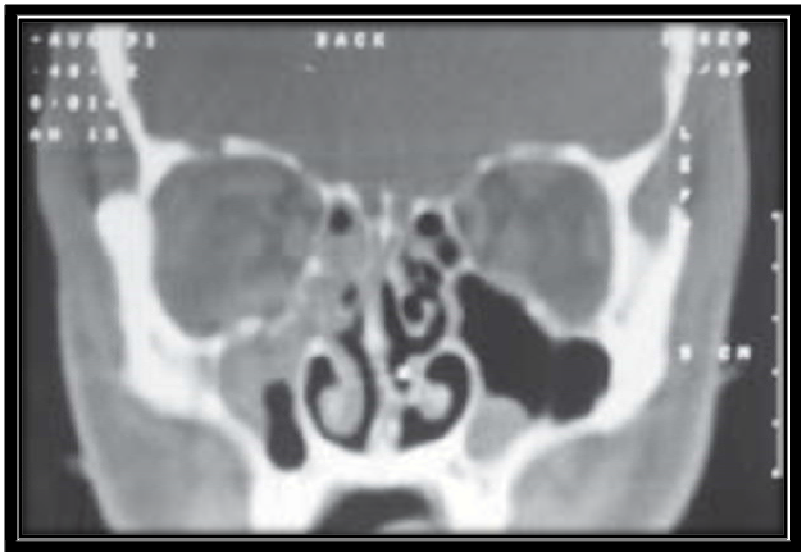


Fundus picture showing optic nerve head avulsion

Fundus picture showing papilledema

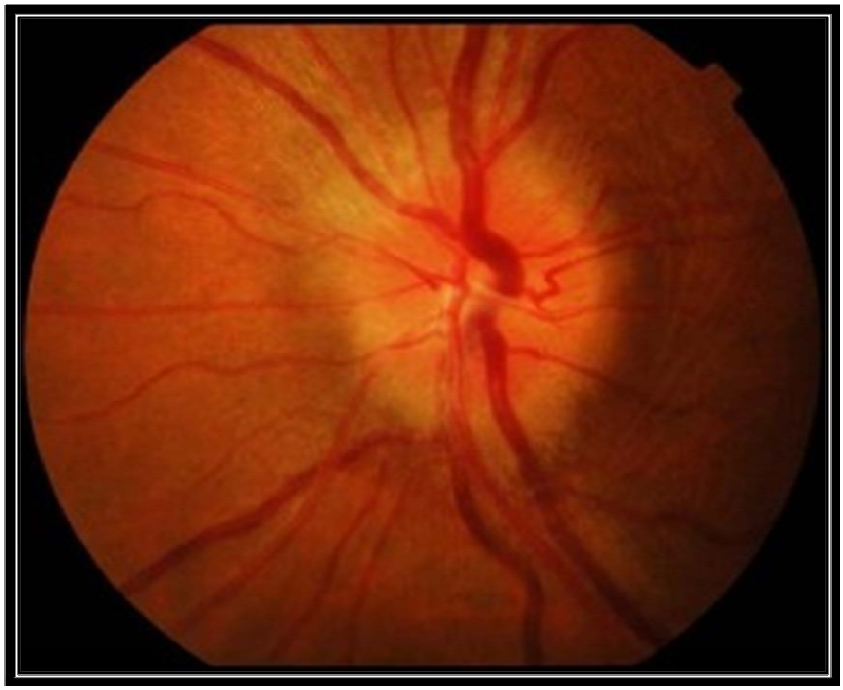
CT scan picture of orbit showing orbital hematoma compressing optic nerve

CT scan picture showing optic nerve sheath hematoma.



CT scan picture showing # floor and medial wall of orbit.

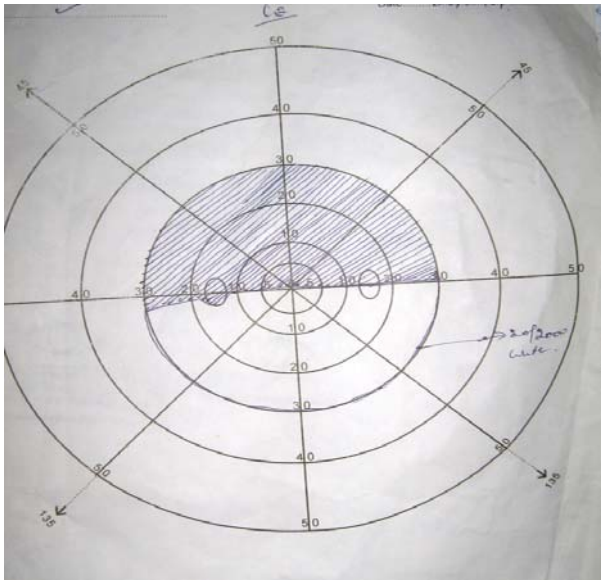
CT scan picture showing optic nerve transection.



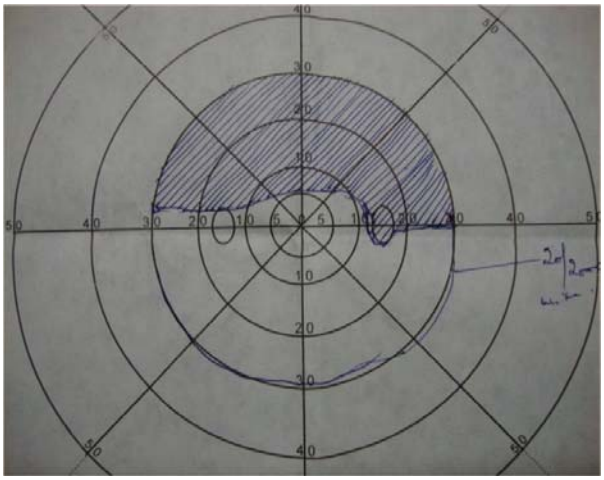
Fundus picture of papillitis

Picture of patient no:58 showing scar over fore head

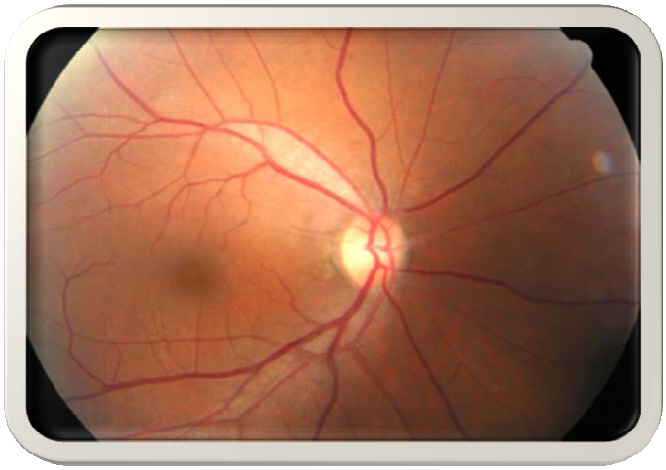
Picture of pt.no:59 with injury over fore head and eyebrow.



Field chart of pt.no:33 showing superior altitudinal defect.

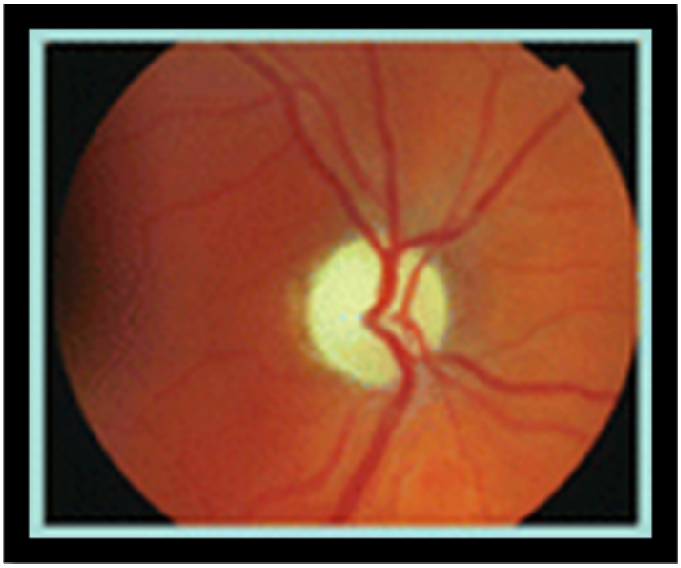


Field chart of pt.no:62 showing superior altitudinal defect.



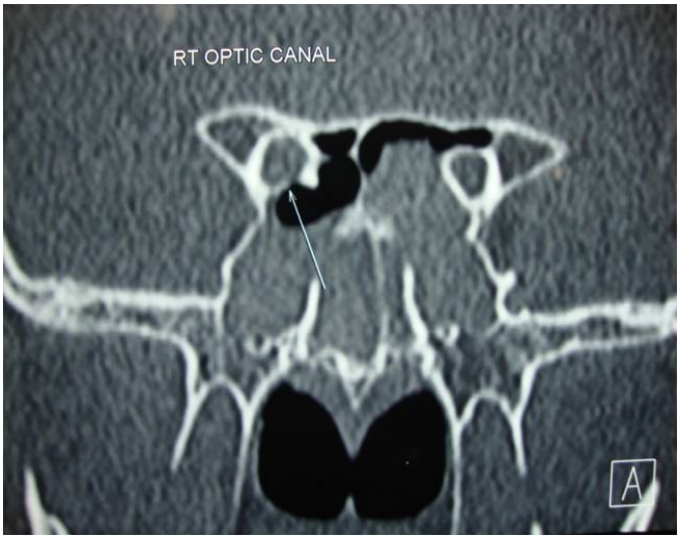
Fundus picture of patient no:65 showing temporal pallor RE

Fundus picture of pt.no:68 showing pallor of the disc



Fundus picture of pt.no:76 showing pallor of the disc

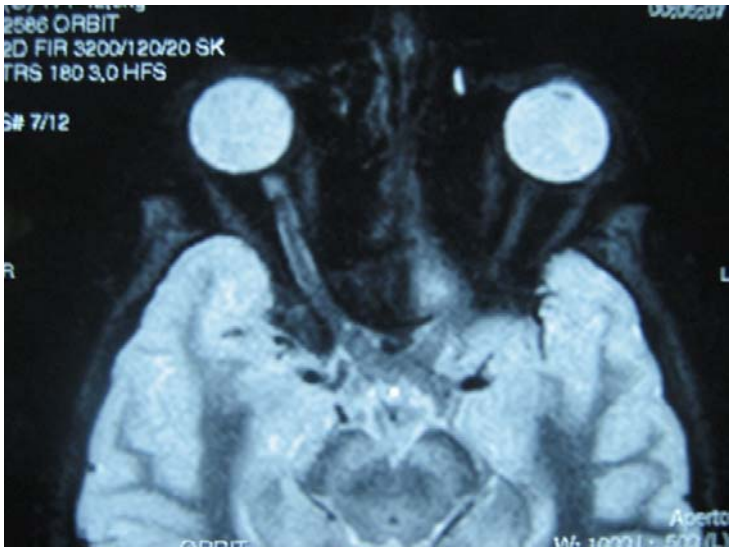
Fundus picture of pt.no:81 showing normal fundus



CT Scan picture of pt.no:62 showing # of medial wall of Rt. Optic canal

CT picture of the same pt. Showing # of lateral wall of Rt. orbit

MRI scan of pt.no:58 showing edema optic nerve



MRI scan of same pt. Edema optic nerve